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PATENT DUCTUS ARTERIOSUS.

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PATENT *ductus arteriosus* is a congenital cardiac lesion due to a failure of the occlusive process at or soon after birth which normally closes this fœtal passage. Its incidence is not infrequent. Maude Abbott,[1] in her series of 1,000 cases of congenital heart disease, found this lesion in 242 cases in all, combined with other anomalies in 150, and as a single defect in 92 cases. It appeared third in frequency in her series, being exceeded by patency of the interauricular and interventricular septa respectively. According to Franklin,[2] Galen first described the patent *ductus arteriosus*, and not Botallo. Chevers,[3] a hundred years ago, reported a case which was confirmed at autopsy. Munro,[4] in 1907, first conceived a surgical approach. Credit should be given to O'Shaughnessy[5] for the first operation for the purpose of closing a patent ductus. The vessel was found to be obliterated, however, and pulmonary stenosis was present. Graybiel, Strieder and Boyer,[6] in 1938, reported an unsuccessful operation on an adult. Gross[7] was responsible for the first successful ligation of a patent *ductus arteriosus*. Since then several series of cases have been reported, and Gross himself has performed more than fifty operations.

Morbid Anatomy.

The size and shape of the patent *ductus arteriosus* vary somewhat. Usually it is approximately from 0·75 to 1·0 centimetre in width and about the same in length. It is not easy to assess the dimensions at operation, as the structure tends to be distorted by the retraction necessary to see it. It is funnel-shaped in most cases, being wider at the aortic than at the pulmonary end. Atheromatous degeneration may be present at the aortic end, but it is more common at the pulmonary orifice and involves the adjacent pulmonary artery. The passage may be represented by a fistula-like opening between the aorta and pulmonary artery. The pulmonary artery is usually dilated. It may equal or exceed the aorta in diameter and may at times reach aneurysmal proportions—for example, three inches in one case.[8] Thinning of the pulmonary arterial wall takes place, rendering it a delicate structure, liable to easy rupture or laceration during operation. The wall of the ductus itself may be exceedingly thin, and in some cases is so thin that it has a bluish, translucent appearance.

In adults, and occasionally in children, a patent ductus may be complicated by subacute bacterial endarteritis, and this, as shown by Maude Abbott, usually involves initially the pulmonary end and the adjacent pulmonary artery. The infection may spread to involve the pulmonary valve, the aorta and the aortic valve. The heart itself is enlarged unless the defect is minimal and the enlargement involves the left ventricle particularly.

The flow through the ductus is arterio-venous—that is, from the aorta to the pulmonary artery. This is due to the fact that the pulmonary pressure is only one-quarter to one-third of that in the aorta. The work of the left ventricle is increased as a result of this aortic leak, which acts like an arterio-venous aneurysm, and the ventricle hypertrophies. The volume of blood circulating through the lungs via the ductus may be very great. In one case[9] it was estimated that while 5·8 litres passed through the right side of the heart, as much as 24·6 litres flowed through the left auricle and ventricle.

Symptoms and Signs.

If the cardiac defect is fully compensated there are no symptoms. The subject appears normal, growth and development are not interfered with and in every respect a normal life may be pursued. This asymptomatic state usually terminates before the age of forty years, although cases have been recorded of patients living much beyond

that period. In the absence of symptoms the murmur is usually discovered in the course of a routine examination, such as a school or insurance examination. In the majority of cases the effects of the lesion are noticeable and may make their appearance in early infancy. The most frequent symptom is easy fatigability and lack of endurance; the child has to lie down, even though the party is not yet over. Other symptoms are retardation of growth, asthenia, a general failure of development, dyspnœa, exhaustion, weakness, and occasionally cardiac pain. Congestive failure may supervene.

By far the most important physical sign is the murmur, which is characteristic and diagnostic in the great majority of cases. The diagnostic feature is its prolongation from the beginning of systole into, and in most cases throughout, diastole. Thus the murmur is described as "continuous", "machinery", "water-wheel", "train-in-tunnel" *et cetera*. Frequently, however, it does not persist until the end of diastole, and a short silent gap may be heard before the first sound. The murmur fades to a variable extent in diastole and is usually distinctly softer than in systole. The point of maximum intensity is in the second left intercostal space close to the sternal border. The murmur is usually propagated widely, and may be audible to the right of the sternum, up to the clavicles, in the axilla and at the pulmonary bases posteriorly. The characteristic murmur is more often present in older children and in adults than in infancy.

In some cases the murmur at the first examination may be purely systolic. If such a patient is examined periodically, the appearance of a diastolic murmur may sometimes be observed, even in so short a period as a few months. This is not likely to occur after childhood. In terminal failure the diastolic and even the systolic element may disappear. This is conceivably due to a lowered aortic blood pressure. Accompanying the murmur there is in most cases a systolic thrill. In a few it may be palpable throughout the cardiac cycle. Its intensity is related to the roughness of the murmur and is correspondingly variable.

There are other signs which are brought about as a result of the arterio-venous leakage—namely, high pulse pressure with low diastolic pressure, pistol-shot sounds along the peripheral arteries and a Corrigan pulse. These are commensurate with the size of the defect and are probably produced by reflex vasodilatation through the sino-aortic reflex, the mechanism being similar to that obtaining in aortic insufficiency.

The radiological signs described by Donovan[10] *et alii* are five, namely: (i) dilatation of the pulmonary artery, (ii) enlargement of the left ventricle with widening of the cardiac shadow, (iii) dilatation of the left auricle, (iv) pulmonary congestion in the mid-zones, (v) "hilar dance". None of these changes is constant. Dilatation of the pulmonary artery and ventricular hypertrophy are usually evident. So-called "hilar dance" is a jerky impulse in the pulmonary arteries seen on fluoroscopic examination. There is also an exaggerated beat of the left ventricle.

It is often stated that the electrocardiograph in patent *ductus arteriosus* shows no abnormality and that axis deviation is absent or slight. Axis deviation, however, may be present, and left axis deviation may be pronounced. Other changes, such as depressed *ST* intervals in Leads II and III and an abnormal *T* wave in Lead IV, may be encountered.

It is an important point that these patients, as they are suffering from an arterio-venous shunt, are not cyanosed, nor do they have clubbing of the fingers or polycythæmia.

Diagnosis.

The diagnosis of patent *ductus arteriosus* is straightforward in most cases, but occasionally may be difficult or even impossible without exploration. The following case record exemplifies this.

A married woman, aged twenty-two years, was admitted to hospital on account of attacks of fatigability, precordial pain and breathlessness, which had caused her to stay in bed for the preceding four months.

On examination the following findings were made. The pulse pressure was raised 110/50/0, pistol-shot sounds were heard, a collapsing pulse was present, and a systolic thrill and continuous murmur were heard in the pulmonary area. The heart was enlarged, and X-ray examination revealed a large left ventricle and a prominent pulmonary artery. The patient was anæmic, and culture from the blood yielded a growth of *Streptococcus viridans*. Leucocytosis and fever were absent. On the basis of a probable patent *ductus arteriosus* with secondary infection, an exploratory operation was performed. A *ligamentum arteriosum* was identified. Autopsy accounted for the foregoing findings by revealing a patent interventricular septum with vegetations upon it and also aortic regurgitation due to subacute endocarditis. The two lesions combined produced a syndrome resembling patent *ductus arteriosus*.

The diagnosis rests upon the presence and location of the characteristic murmur, supported by some or all of the signs previously outlined. Patent *ductus arteriosus* must be differentiated from other congenital anomalies. Those lesions associated with veno-arterial shunts are at once excluded by the presence of cyanosis. Patent interventricular septum is recognizable by the purely systolic murmur in the region of the third left intercostal space. This lesion may coexist with a patent ductus. A defect of the interauricular septum may somewhat resemble a patent ductus. The X-ray films show a similar prominence of the pulmonary artery. Thrill and murmur may be absent altogether, but on the other hand they may both be present and are then found in the pulmonary area. The murmur is systolic, but there may be a diastolic element too, due to pulmonary incompetence. This murmur, however, when present, does not resemble that of a patent ductus. A further differential feature is the electrocardiographic tracing, which reveals right axis deviation. Coarctation of the aorta is distinguished by brachial hypertension, and by the brachial pressure being greater than the popliteal pressure. The common classical signs of this lesion, such as rib erosion and prominent arterial anastomoses, may be lacking in a child. Pulmonary stenosis gives rise to a systolic murmur, and usually other defects are present, such as patent interventricular septum and right ventricular hypertrophy. A continuous venous hum may be heard in the neck and conducted to the chest in some children. It can be obliterated by compression of the neck veins.

Prognosis.

The prognosis in patent *ductus arteriosus* is poor, despite the fact that some patients reach the fifth decade, and in a few isolated instances even the seventh decade. In Abbott's 92 cases the average age at death was twenty-four years, death being due to heart failure in 40 and to bacterial endarteritis or endocarditis in 21. Keys and Shapiro,[11] analysing 60 cases among adults, found that 40% died of infection and 28% of congestive failure. They estimated that at the age of seventeen years the presence of a patent ductus reduced the average expectation of life by twenty-five years. Bullock *et alii*[12] have analysed 80 proved cases, all the patients being over the age of three years. By the age of fourteen years 14% had died, by the age of thirty years 50% had died, and by the age of forty years 71% had died. It is the opinion of some authorities that these autopsy statistics may be weighted by the fact that those patients in whom infection or decompensation develops are more likely to be brought to hospital and to autopsy. On the other hand, it is remarkably unusual to find this lesion in a mature adult in the general course of clinical practice or in the conducting of routine examinations, such as "medical boarding" for the services.

Treatment.

Since 1939 patent *ductus arteriosus* has been treated by ligation or by division with suturing of the divided ends. The latter technique was developed by Gross[12] and is now utilized by him as a routine measure. The operation is not without risk, as the ductus and pulmonary artery are very thin-walled structures. This fact has been responsible for the most frequent and dramatic cause of death—namely, fatal hæmorrhage. Shapiro found that in 137 operations this accident occurred in eleven cases.

Indications for surgical interference are: (i) subacute bacterial endarteritis or endocarditis, (ii) cardiac insufficiency and cardiac hypertrophy, (iii) mental and physical retardation. In the case of infection, operation has resulted in apparent cure in about two-thirds of the reported cases. Touroff[10] has shown that in such cases samples taken from the peripheral blood as soon as forty minutes after operation have failed to yield a growth of micro-organisms. It is thought that the infection dies out because venous blood provides a less favourable milieu for the organisms than a mixture of venous and arterial blood. The reduction of intraarterial pulmonary pressure and the filtering action of the pulmonary capillary bed are probably additional factors influencing the outcome.

Surgical closure of the ductus is indicated when the shunt is great, as evidenced by cardiac hypertrophy, low diastolic arterial pressure, high pulse pressure, pistol-shot sounds and intolerance of exertion. These signs denote cardiac embarrassment and the probability of subsequent failure.

The question as to whether patients with fully compensated patent *ductus arteriosus* should be submitted to operation is an open one and must remain so until the final results of operation can be more comprehensively surveyed and until the minimum operative mortality rate is reached. In the decision to pursue a conservative form of treatment, it is to be considered that, at least on American figures, about one affected person in three is liable to bacterial infection. Furthermore, even a small arterio-venous communication places an undue burden upon the circulation, and persons who have undergone the operation experience new vigour and energy even though they were not conscious of a lack of it beforehand. At present it would seem advisable to defer operation when growth, intellect, heart size, exercise tolerance and physical endurance are normal. It may be stated that these cases are in the minority.

Reports of Cases.

CASE I.—A schoolgirl, aged six years, was referred to the hospital in July, 1941, by the Department of Public Instruction because of a cardiac murmur. There was a history of frequent coughs and colds and of failure to gain in weight. She was pale and of asthenic build. On examination of the child, the apex beat was two inches from the mid-line in the fourth intercostal space. A faint systolic thrill was felt over the pulmonary area. A loud, rough murmur, which was almost, but not quite, continuous, was audible with maximum intensity in the second left intercostal space. No other murmurs were present. The systolic blood pressure was 100 millimetres of mercury and the diastolic pressure 50. X-ray examination revealed slight widening of the base of the cardiac shadow and prominence of the pulmonary artery. The electrocardiogram was normal. The red blood cells numbered 4,200,000 per cubic millimetre and the leucocytes 6,700 per cubic millimetre.

The child was kept under observation for two years, but her condition failed to improve. Her weight was stationary at three stone and three-quarters of a pound. Therefore it was decided to recommend her for operation. On July 15, 1943, the operation was successfully performed, despite considerable hæmorrhage from the *ductus arteriosus*. After operation her convalescence was satisfactory. The murmur and thrill had disappeared. The systolic blood pressure was 80 millimetres of mercury and the diastolic pressure 45. The child became more energetic. She gained twelve pounds in weight and three inches in height in the ensuing eighteen months. Further examination after that interval showed that the systolic blood pressure was 75 millimetres of mercury and the diastolic pressure 30. No murmurs were present. There was little reduction radiologically in the size of the heart, however, and the pulmonary artery prominence was not diminished. She was well in every way, was enjoying an outdoor life and had figured as a winner in a sprinting event.

This patient was the first in this country to be operated upon for patent *ductus arteriosus*. The operation was performed by Dr. B. T. Edye.

CASE II.—A schoolgirl, aged seven years, whose identical twin sister also suffered from congenital heart disease, was first examined in 1941 on account of a persistent elevation of temperature. A loud murmur had been discovered on routine examination at the age of four years. The child suffered from periodic bouts of low-grade fever and was susceptible to respiratory infections. Her chief symptom was the premature onset of fatigue during the day's activities, which would necessitate her resting. She had been kept under observation for two years, and during this time symptoms became more pronounced. In June, 1943, examination showed her to be a healthy looking girl, well grown and well formed for her age. Her weight was four stone twelve pounds. The systolic blood pressure was 105 millimetres of mercury and the diastolic pressure 0, a slight dulling of sound being observed at the 35 millimetre mark. A systolic bruit was audible along the brachial artery. The heart was much enlarged and the cardiac action was augmented. The apex beat was four inches from the mid-line in the fifth intercostal space (the mid-clavicular point was at two and three-quarter inches). A systolic thrill was present over the second and third left intercostal spaces. At the pulmonary area there was a continuous murmur, rough and loud during systole, somewhat softer during diastole, and conducted widely over the thorax anteriorly and posteriorly. The red cells numbered 3,480,000 per cubic millimetre, the hæmoglobin value was 72%, and the total leucocytes numbered 4,050 per cubic millimetre, 22·5% being neutrophile cells. No immature cells were seen. X-ray examination showed the heart to be considerably enlarged owing to left ventricular hypertrophy. There was a convexity of the left border corresponding to the position of the pulmonary artery. Mottled congestion was present in the pulmonary midzones. The electrocardiogram revealed no abnormality.

On November 19, 1943, the ductus was ligated. The thrill and murmur were abolished and the blood pressure readings immediately after operation were 115 millimetres of mercury, systolic, and 55, diastolic. Convalescence was uneventful. The child was last examined in February, 1945, fifteen months after operation. The systolic blood pressure was 115 millimetres of mercury and the diastolic pressure 55. No murmurs were present. Radiologically the heart was smaller. The transverse diameter was reduced by more than a centimetre, but the pulmonary artery dilatation persisted, and the left ventricle was still somewhat prominent. The lung fields were clear. She had lost her tendency to respiratory infections, her weight had increased to five stone eleven pounds and she had an abundance of tireless energy.

CASE III.—A schoolboy, aged ten years, was referred to the hospital with a history that two years previously he had become listless and fatigued and was always wanting to lie down. He had no energy and was disinclined to go to school. A psychologist was consulted and he discovered a heart murmur. The boy's lassitude continued. The school doctor had stopped him from drilling, because it "knocked him up". He was unable to carry any loads, and in hot weather particularly he was lethargic and inclined to drag his feet when walking. He was not backward in growth, development or school attainment.

On examination of the patient, his physique and nutrition were normal for his age. His weight was five stone twelve pounds and his height four feet eight inches. The systolic blood pressure was 105 millimetres of mercury and the diastolic pressure 50. The heart was enlarged. The apex beat was three inches from the mid-line in the fifth left intercostal space, and was forcible and diffuse. A systolic thrill was palpable in the pulmonary area. A blowing systolic murmur was present at the apex. At the pulmonary area a continuous murmur was present, harsh in systole, fading in diastole. The second pulmonary sound was accentuated. An electrocardiogram showed no abnormality. X-ray examination of the heart revealed no abnormality apart from some prominence of the left ventricle. No pulmonary congestion was present.

On March 30, 1944, a patent *ductus arteriosus* was ligated through a postero-lateral approach with resection of the fourth rib. Convalescence was somewhat retarded, owing to the development of pneumothorax on the right side and pneumonia with pleural effusion on the left. The condition ran a febrile course for three weeks. Resolution was slow, but the lungs were quite clear radiologically after two months.

Ten months after operation he was in excellent health and he had gained over a stone in weight and had grown three inches taller. The systolic blood pressure was 95 millimetres of mercury and the diastolic pressure 45. The apex beat was three inches from the middle line, in the mid-clavicular line. No murmur was present. X-ray examination showed the heart to be narrower and less globular in shape than previously. The boy had recently been the winner of a swimming race.

CASE IV.—A girl, aged four years, was referred to the hospital with a view to operation because of considerable dyspnœa on slight exertion, such as walking, and some retardation of growth and development. The mother stated that "she seems to run around for a while and then stands puffing and blowing before she can get her wind again".

On examination, the child was pale and somewhat undersized for her age. The systolic blood pressure was 100 millimetres of mercury and the diastolic pressure 0. A systolic thrill was palpable at the second left intercostal space. At the apex a systolic murmur was present which was conducted towards the axilla. At the pulmonary area a murmur, continuous throughout systole and diastole, was present and was propagated along the carotid arteries and was also audible at the base of the left lung. It was a typical machinery murmur. A blood count showed that the red cells numbered 3,900,000 per cubic millimetre, and that the hæmoglobin value was 70%. The leucocytes were normal in number and type. An electrocardiogram showed sinus arrhythmia. X-ray examination revealed considerable enlargement of the heart, mainly involving the left ventricle, convexity of the left border due to pulmonary artery dilatation, and also fairly well-marked congestion of the pulmonary mid-zones. In the right oblique film, slight dilatation of the left auricle was seen.

Operation was performed on April 13, 1944, through the postero-lateral approach with resection of the fourth rib. The patent ductus was doubly ligated. Immediately afterwards the blood pressure was 120 millimetres of mercury, systolic, and 70, diastolic. The patient's condition was fairly satisfactory for the ensuing twenty-four hours, although the pulse rate ranged between 140 and 150 per minute. During the next twelve hours it rose to over 170 per minute, and the child rapidly became distressed and died. Post-mortem examination revealed pulmonary œdema.

CASE V.—A waitress, aged twenty years, was referred from another State with a view to operation. She gave a history of having been "short of wind" since she was a child. She complained of palpitation of the heart when excited or after exercise. She was dyspnœic on exertion. She complained that she became fatigued very easily. She was not specially prone to respiratory infections, but when they occurred recovery was very slow.

On examination, the patient appeared to be a well-nourished young woman with good physical development. She was pale. The systolic blood pressure was 120 millimetres of mercury and the diastolic pressure 65. The heart was enlarged. The apex beat was five inches from the middle line in the fifth left intercostal space. A pronounced thrill was palpable in the second and third left intercostal spaces at the sternal border. A systolic murmur was present at the mitral area. In the second left intercostal space a continuous harsh murmur with increment during systole was heard. The murmur was propagated along both carotid arteries, and into the left axilla, and could be heard at the bases of both lungs. A blood count revealed no anæmia. The hæmoglobin value was 78%, and the leucocytes numbered 8,200 per cubic millimetre. An electrocardiogram showed left axis deviation. X-ray examination showed the heart shadow to be considerably enlarged owing to left ventricular hypertrophy. The cardio-thoracic ratio was 3:5. There was a definite but not pronounced convexity of the left border of the heart due to pulmonary artery dilatation. Pulmonary congestion was not evident.

On April 20, 1944, operation was undertaken through the postero-lateral approach with resection of eight inches of the fourth rib. The ductus was identified, and during manipulation hæmorrhage occurred owing to a tear in the ductus itself. This was incompletely controlled, and proved fatal after about thirty-six hours.

CASE VI.—A girl, aged three years, was brought to the hospital on December 7, 1943, on account of enlargement of the heart, which had been discovered one year previously. One week before she had had an attack in which she fainted and went blue. It lasted only a few minutes and she quickly recovered. She was otherwise quite well except for frequent attacks of bronchitis, from one of which she was recovering at the time of examination. The child's mother stated that she was not dyspnœic, but that she would not walk a great distance.

On examination, the child appeared stunted and grossly underweight. Her weight was one stone ten and a half pounds. Her chest was "rickety", with a depression in the region of the *manubrium sterni*, and there was a bulge to the left of the sternum. The cardiac impulse was located in the fourth left intercostal space, two and a half inches from the middle line; it was diffuse and forcible. A systolic thrill was palpable over the carotid arteries. A thrill was faintly palpable in the pulmonary area. A to-and-fro murmur was audible at the cardiac apex. In the second left intercostal space a loud, harsh murmur limited to systole was present. An electrocardiogram showed prominent *Q* waves and depressed *S-T* intervals in Leads II and III with diphasic *T* waves in Lead III. The report on the electrocardiogram was "myocardial defect". X-ray examination revealed considerable enlargement of the heart. The cardio-thoracic ratio was 10:17. The left ventricle was prominently enlarged to the left and downward. The left border of the heart above the ventricle was concave, and there was no evidence of pulmonary artery dilatation. There was considerable mottling of the lungs in the region of the hila, indicative of vascular congestion. A blood count showed that the red cells numbered 4,200,000 per cubic millimetre, the hæmoglobin value was 75%, and the white cells numbered 8,450 per cubic millimetre. A provisional diagnosis of patent interventricular septum was made.

On June 13, 1944, the child was reexamined. She had failed to thrive. The systolic blood pressure was 95 millimetres of mercury and the diastolic blood pressure 0; the pulse rate was 130 per minute. The heart action was greatly augmented. The apex beat was half an inch beyond the mid-clavicular point. A basal thrill was present to the left of the sternum and was widely propagated. The murmur at the pulmonary area was rough and harsh and extended into diastole, although it faded off to a silent interval before the first sound. The electrocardiogram was unchanged. X-ray examination revealed further enlargement of the heart and increased congestion of the lungs. The left border of the heart had become less concave. Pistol-shot sounds were audible over the peripheral arteries. A diagnosis of patent *ductus arteriosus* was made.

FIGURE I.
Case VI. Patient before operation.

FIGURE II.
Patient seven months after operation; she had gained 12 pounds in weight.

On June 24 ligation of the patent ductus was performed through the anterior approach. The post-operative course was uneventful. On February 7, 1945, the child was examined again. She had maintained greatly improved health and vigour, and had been free of respiratory infections. Her weight was two stone twelve pounds. She had gained twelve pounds in the last seven months. The systolic blood pressure was 80 millimetres of mercury and the diastolic pressure was 45. There was no systolic arterial bruit. The apex beat was two and a quarter inches from the middle line and was situated in the mid-clavicular line. The heart's action was not accentuated. The only murmur present was a slight systolic murmur audible at the aortic and pulmonary areas. X-ray films showed the heart to be reduced in size and the pulmonary fields clear.

CASE VII.—A housewife, aged thirty-five years, was admitted to the hospital on July 13, 1944, complaining that she had always been dyspnœic on exertion and had felt tired. For ten weeks she had suffered from attacks of pain in the precordium, brought on by exertion. The pain occurred on an average three times in the day, was of a dragging nature and lasted from two or three to ten minutes. It never came on while she was at rest, and lying down relieved it. She stated that the heart beat quickly and thumped during the attacks of pain. She had considerable breathlessness on exertion. Although she lived in a tiny flat she had great difficulty in performing her duties, and finally was forced to curtail them on account of the anginal attacks. She had two children, aged nine years and six years respectively. At the age of five years she had been an in-patient at the Royal Alexandra Hospital for Children on account of nephritis; no note of any cardiac signs was made in the history.

On examination, the patient was a healthy looking woman. Her weight was eight stone ten pounds. The pulse rate was 88 per minute. The systolic blood pressure was 180 millimetres of mercury and the diastolic pressure 100. Her complexion was somewhat sallow. A systolic thrill was palpable in the second left intercostal space and slightly palpable in the third left intercostal space. The thrill was not pronounced. The apex beat was four inches from the middle line in the fifth left intercostal space, the mid-clavicular point being at three and a half inches. A systolic murmur was present at the apex. At the pulmonary area a typical ductus murmur was present, continuous and conducted somewhat towards the left shoulder and the sternum, not elsewhere. With a Bowles stethoscope the diastolic component was rather high-pitched, and below the manubrium it had a whining tone. Electrocardiographic examination showed sagging of the *S-T* intervals in Leads II and III. The report on the electrocardiogram was "marked myocardial defect". X-ray examination revealed considerable left ventricular hypertrophy with marked prominence on the left border, due to a dilated pulmonary artery. The cardio-thoracic ratio exceeded 1:2. In the right oblique film no dilatation of the left auricle could be seen. The lungs were congested in the hilar region. A blood count showed that the red cells numbered 5,000,000 per cubic millimetre, and the hæmoglobin value was 91%.

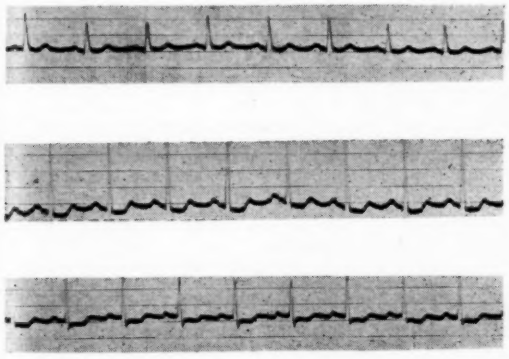

FIGURE III.
Case VII. Note changes in *S-T* interval in Leads II and III.

Operation was performed on July 15 through the second left intercostal space anteriorly, and the ductus was ligated. The post-operative period was uneventful, and the patient was discharged from hospital fifteen days later. On February 7, 1945, seven months after operation, she stated that her exercise tolerance was excellent. She was able to dance and climb hills without any pain or dyspnœa. Her weight was eight stone eight pounds. The systolic blood pressure had fallen to 135 millimetres of mercury and the diastolic pressure to 95. No thrill or murmur could be detected. The first pulmonary sound was reduplicated and the second was considerably accentuated. The electrocardiographic changes had returned towards normal, the *S-T* intervals being less depressed. The *T* wave in Lead IV was flattened. X-ray examination of the heart showed the total area to be smaller and the transverse diameter to have been reduced by a centimetre. There were no signs of congestion in the lungs.

CASE VIII.—A schoolgirl, aged ten years, was referred by another hospital with a view to operation for a patent ductus. At the age of seven years she had been found to have "a bad heart". She had been fairly well until two months previously. Since then she had complained of lack of energy and of tiring easily. She had suffered from dizzy turns, about six per day, which lasted for a minute or so. She had been breathless on exertion for the same period. She had a tight feeling in the precordium, which lasted about an hour after effort. She suffered from severe throbbing of the heart after slight exertion. Her weight was five stone eight pounds, her height four feet six inches, and she was a pale, asthenic-looking girl. The systolic blood pressure was 120 millimetres of mercury and the diastolic pressure 65. No thrill was palpable. The apex beat was three inches from the middle line in the fifth left intercostal space in the mid-clavicular line. A systolic murmur, which was not conducted, was present at the apex. At the pulmonary area in the second left intercostal space a rough, continuous murmur was audible throughout the cardiac cycle, louder during systole. A blood count showed that the red cells numbered 4,500,000 per cubic millimetre, the hæmoglobin value was 79% and the leucocytes numbered 8,550 per cubic millimetre. The electrocardiogram revealed no abnormality. X-ray examination showed that the heart was not greatly enlarged. The transverse diameter was four inches and the cardio-thoracic ratio was 4:9. The left ventricle was slightly prominent, but there was a pronounced bulge on the left border of the cardiac shadow due to dilatation of the pulmonary artery. Slight mottling of the middle zones of the lungs near the hila could be seen.

Operation was undertaken on August 12, 1944, by the anterior approach through the second left intercostal space, and the patent *ductus arteriosus* was ligated. The post-operative course was somewhat retarded on account of pleural effusion. At a follow-up examination six months later the following observations were made. The child was reported to be "real well". She stated that she could run, which she had not been able to do before, and that she no longer had throbbing or breathlessness on exertion. Her weight was six stone, a gain of six pounds. Her height was four feet nine inches. The systolic blood pressure was 115 millimetres of mercury and the diastolic pressure 65. The apex beat was three and a half inches from the middle line. No murmurs were present.

CASE IX.—On May 2, 1944, a female child, aged three years, was brought to the hospital by her guardian because she was not eating well and was not gaining in weight. She was energetic, but tired easily. On examination of the child, physical development was obviously poor, her weight being 21 pounds. Her colour was pale. Her expression was bright and alert, but instead of being able to form sentences she was capable of saying only a few words, such as "daddy". Fairly pronounced deformity of the anterior thoracic wall was present, with pigeon-chest formation. Harrison's sulcus was present also. The systolic blood pressure was 70 millimetres of mercury and the diastolic pressure 0. The apex beat was palpable in the fifth left intercostal space, just beyond the mid-clavicular line. A pronounced systolic thrill was present over the pulmonary area. In the second left intercostal space a loud harsh murmur, which was almost continuous, could be heard. It was propagated to the apex, to the axilla and aortic areas, and also to the bases of the lungs posteriorly. Pistol-shot sounds were audible over the brachial and femoral arteries. An electrocardiogram failed to reveal axis deviation, but the *T* waves were abnormal in that in Leads II and III they were of greatly increased amplitude—ten millimetres—and were peaked. X-ray films of the heart showed it to be globular in shape, with enlargement of the right auricle and pronounced dilatation of the pulmonary artery. The results of a blood count were as follows: the red cells numbered 4,500,000 per cubic millimetre, the hæmoglobin value was 66%, and the white cells numbered 8,500 per cubic millimetre. Thoracotomy was deferred until September 16, 1944, on account of persistent head cold and cough, and of chronic nasal catarrh, which was treated by adenoidectomy. On that date operation was undertaken through the second left intercostal space and the patent ductus was ligated. The post-operative course was uneventful, and the child was discharged from hospital fourteen days later.

Examination five months later gave the following findings. The child's appearance was healthy, she was bright and active, and she was now able to make short sentences. Her appetite was satisfactory and she had no fatigue. Her weight was 26·5 pounds—an increase of five pounds. Pigeon

chest was still present. The systolic blood pressure was 70 millimetres of mercury and the diastolic pressure 35. There was no thrill. The apex beat was still slightly displaced, being two and a quarter inches from the middle line (one-quarter of an inch beyond the mid-clavicular line). X-ray examination showed that the heart was reduced in its total size, and that although the child's weight had increased by five pounds and she was five months older, the transverse diameter of the heart was reduced from 3·75 inches to 3·25 inches. The congestion in the lung fields had disappeared. The abnormal *T* waves in the electrocardiogram underwent a considerable change in their character towards normal within three days after operation.

The most satisfactory thing in this case was the mental improvement.

CASE X.—This girl, aged eight years, was the identical twin sister of the patient in Case II. She was first examined in 1941, and at that time a diagnosis of patent interventricular septum was entertained on the basis of a rough, purely systolic murmur in the second and third left intercostal spaces. She had been kept under observation and was again examined on August 16, 1943. At that time her only symptoms were breathlessness on exertion of a few months' duration and some lack of vitality. She was a well-grown girl with no physical or mental retardation. The pulse rate was 96 per minute, and the systolic blood pressure 102 millimetres of mercury and the diastolic pressure 50. The apex beat was three inches from the middle line. At the mitral area were heard a systolic and a short diastolic murmur. At the base of the heart a rough systolic murmur was present, maximal in the third left intercostal space. The blood count showed that the red cells numbered 3,500,000 per cubic millimetre, the hæmoglobin value was 77% and the leucocytes numbered 8,400 per cubic millimetre. An electrocardiogram showed slight left axis deviation. X-ray films of the heart showed that it had increased considerably in size since it had first been radiologically examined three years before. This enlargement was due almost entirely to left ventricular hypertrophy. The cardio-thoracic ratio exceeded 1:2. There was slight mottling of the lungs due to increased vascularity. The left border of the heart was concave, showing no evidence of pulmonary artery dilatation. The diagnosis of patent interventricular septum was maintained.

On reexamination of the patient in December, 1943, the physical findings were essentially unaltered. In June, 1944, the child's mother complained that whereas the twin sister, who had been successfully operated upon, had no respiratory embarrassment and was charged with an abundance of energy, this child had become more breathless, listless and disinterested. Her condition simulated that of her sister before operation. On auscultation of her heart, it was found that the basal murmur had changed. The murmur, which formerly had been purely systolic, was now continuous beyond the second sound, and could be heard throughout the greater part of diastole.

The patient was admitted to hospital on October 23, 1944, with a view to operation. Some pallor was noted. The pulse rate was 76 per minute. The systolic blood pressure was 110 millimetres of mercury and the diastolic pressure 55, but the bruit could be heard faintly down to zero. A faint systolic thrill was present at the base of the heart to the left of the sternum. The apex beat was forcible and was three and a half inches from the middle line. A blowing systolic murmur was present at the mitral area. In the second left intercostal space a murmur was heard which was continuous except for a slight break at the end of diastole and immediately preceding the first sound. It was propagated to the left clavicle, to the aortic area and posteriorly. An electrocardiogram showed slight left axis deviation. A review of the X-ray films showed still further enlargement of the heart, involving both the left ventricle and the right auricle. The left border still showed no sign of convexity, such as is observed with pulmonary artery dilatation. On October 28 operation was undertaken by way of the anterior approach, and the ductus was identified and ligated. While the second ligature was being tightened, however, the ductus tore at its junction with the aorta. All attempts at hæmostasis were unsatisfactory, owing to the friability of the structures. The patient died two and a half hours after leaving the operating theatre.

CASE XI.—A married woman, aged twenty-four years, was referred to the hospital with a diagnosis of patent *ductus arteriosus* with a view to operation. Her complaints were that she had always been inclined to be breathless on exertion and had noted tiredness after exertion. Her activities had always been reduced. She had had cramp in the left arm and precordium for three months. Sixteen months previously she had had her first child; during the pregnancy she was well. For the last three months, however, her symptoms had increased, and she had been unable to do housework. Effort caused her to suffer from a feeling of a dragging weight in the precordium, which travelled down the left arm as a cramp-like pain. It was not extremely severe, yet made her afraid to move and at times gave her a feeling of extreme lethargy and generalized weakness.

On examination, the patient was normally developed. The pulse rate was 92 per minute, and the systolic blood pressure was 145 millimetres of mercury and the diastolic pressure 65. Arterial pulsation was present above the sternum and in the right side of the neck. A systolic bruit was audible over the brachial artery. A coarse thrill was palpable over the base of the heart on the left side and also suprasternally. The apex beat was four and a half inches from the middle line in the fifth left intercostal space and was beyond the mid-clavicular line. It was forcible and sustained. At the pulmonary area a harsh machinery murmur was heard throughout the cardiac cycle; it obscured the first and second sounds. There was little diminution during diastole. The murmur was propagated widely and could be well heard below the left scapula. The aortic second sound was increased. A systolic murmur was present at the mitral area. A blood count showed that the red cells numbered 4,000,000 per cubic millimetre, the hæmoglobin value was 88% and the white cells numbered 6,000 per cubic millimetre. X-ray examination in the postero-anterior position revealed slight prominence of the left ventricle, and also a convexity on the left border of the heart indicative of a dilated pulmonary artery. The report on the electrocardiogram was "right axis deviation"; this was of slight degree. In the right oblique film no dilatation of the left auricle could be made out. The patient was treated by bed rest for a period of six weeks. Although apprised of the operative risk and of the previous fatality, she demanded operation, as she was unable to continue as before. Accordingly, operation was undertaken on November 9, 1944, under basal "Avertin" and cyclopropane anæsthesia. After about forty minutes' anæsthesia the pulse rate became rapid, reaching 140 per minute, and the patient's colour deteriorated. Nitrous oxide and oxygen were substituted for cyclopropane, and one millilitre of "Digoxin" was given, 0·5 millilitre intravenously and 0·5 millilitre intramuscularly. Her condition thereafter appeared to be satisfactory. The ductus, when isolated, was found to be short and funnel-shaped; indeed, it appeared as though the pulmonary artery was caught up and attached to the aorta. At its junction with the aorta it was about a centimetre in width. Double ligation of the ductus caused the diastolic pressure to rise from 40 to 90 millimetres of mercury. The thrill disappeared. On auscultation with a sterile stethoscope placed on the ligated vessel, a constant murmur, low, distant and uniform, without much systolic increment, could be heard. Twenty minutes after ligation the diastolic pressure was noted to have risen a further 20 millimetres of mercury to 110, and the systolic pressure had fallen from 180 to 160 millimetres of mercury. Ten minutes later the figures were 140 millimetres of mercury, systolic, and 110, diastolic. The pulse rate had not increased. The operation was concluded and the anæsthesia was discontinued. Suddenly the patient's colour became cyanotic and after a few gasps she ceased breathing. The pulse was imperceptible and heart sounds were absent. Attempts at resuscitation by the intracardiac injection of adrenaline and by artificial respiration with oxygen were of no avail.

In the circumstances this became a coroner's case, and unfortunately no autopsy was performed.

CASE XII.—A boy, aged four years, was referred to the hospital with a diagnosis of patent *ductus arteriosus*. The murmur had been discovered on medical examination three months previously. The mother complained that he cried a great deal and that his appetite was unsatisfactory. He was bad-tempered and spiteful. The mother said that he was failing seriously. Dyspnœa was not present.

On examination, the patient was a pale, miserable, poorly nourished and under-developed boy weighing 27·5 pounds (one stone underweight). An almost continuous thrill was readily felt in the second and third intercostal spaces. The systolic blood pressure was 110 millimetres of mercury and the diastolic pressure 45. The apex beat was two and a half inches from the middle line in the fifth left intercostal space, half an inch outside the mid-clavicular line. A harsh, loud, continuous, machinery murmur was audible in the second left intercostal space and was conducted widely. It could be heard above the left clavicle, at the aortic area and in the region of the left scapula. X-ray examination revealed broadening of the heart shadow with prominence of the left ventricle, as well as some dilatation in the region of the

right auricle. There was no evidence of left auricular dilatation in the right oblique film. The transverse diameter of the heart was 3·75 inches; that of the thorax was 6·5 inches. There was no evidence of pulmonary artery dilatation, the left border of the heart being concave below the aorta. Mottling of the lungs was present due to increased vascularity. A blood count revealed that the red cells numbered 4,100,000 per cubic millimetre, the hæmoglobin value was 83% and the leucocytes numbered 8,400 per cubic millimetre. An electrocardiogram revealed no abnormality.

Operation was undertaken on December 14, 1944, through the anterior approach, and the patent *ductus arteriosus* was doubly ligated. The post-operative period was uneventful. Two months after operation the child's health and well-being were greatly improved. His weight had increased by one pound to 28·5 pounds. The systolic blood pressure was 70 millimetres of mercury and the diastolic pressure 25. No murmur could be detected. The apex beat was two inches from the middle line in the fourth left intercostal space.

In this case the interval after operation was not sufficiently long for a satisfactory follow-up study.

CASE XIII.—A girl, aged fourteen years, was referred to the hospital on March 6, 1945, with a provisional diagnosis of patent *ductus arteriosus*. She had led an active and more or less normal life. At intervals of some months she had had "attacks" characterized by palpitation, dyspnœa, pulsation in the neck, fever and pain in the chest. These episodes were so severe that she had to be admitted to the local hospital. She was breathless on mounting steps or climbing hills. The menarche had failed to appear. The previous illnesses and family history were irrelevant.

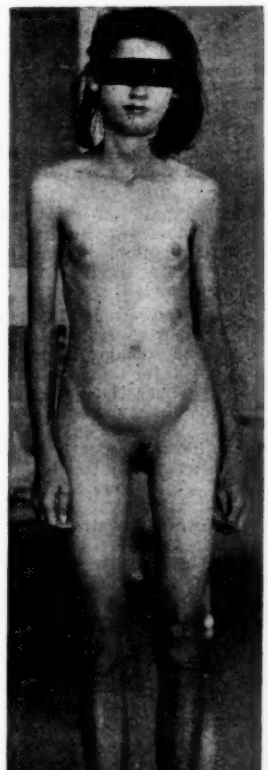

FIGURE IV.

Case XIII. A girl, aged fourteen years, showing poor physical development; weight, four stone ten pounds.

On examination, the patient was pale and poorly developed (Figure IV). Her height was four feet nine inches and her weight four stone ten pounds. The systolic blood pressure was 130 millimetres of mercury and the diastolic pressure 44. The cardiac action was greatly augmented and the impulse was forcible and diffuse. The apex beat was three and a half inches from the middle line, three-quarters of an inch beyond the mid-clavicular line. A continuous thrill was palpable at the pulmonary area. At the apex it could be felt only during systole. It was conducted along the carotid arteries. A continuous roaring murmur was present, maximal in the second left intercostal space and propagated widely over the thorax. The blood count gave normal results, and attempted culture of micro-organisms from the blood was unsuccessful. An electrocardiogram showed depression of the *S-T* segment in Lead I with a low take-off, a diphasic *T* wave in Lead I, and elevation of the *S-T* segment in Lead III with a high take-off. Considerable left axis deviation was present. X-ray examination revealed enlargement of the left ventricle and left auricle, straightening of the left border of the heart and congestion of the middle zones of the lungs. Hilar dance was not present.

Operation was undertaken for the purpose of ligation of a patent ductus. This structure was found to be very large and to exceed in size the others in this series. It was doubly ligated, and at operation the murmur was obliterated, the tied duct being auscultated with a sterile stethoscope before closure of the mediastinum. The post-operative period was uneventful.

CASE XIV.—A girl, aged seven years, was brought to the hospital in November, 1943, with a history of breathlessness on slight exertion, poor nutrition, difficulty with her meals and frequent respiratory infections. One year previously at another hospital the parents had been told that she had heart disease. On examination, she was pale and asthenic, although not grossly underweight. Her weight was three stone seven pounds and her height four feet two inches. The systolic blood pressure was 170 millimetres of mercury and the diastolic pressure 0, the sound being audible down to zero. A Corrigan's pulse was present, and sharp systolic sounds were heard along the brachial and femoral arteries. Pulsation was present in the epigastrium and pronounced in the carotids in the neck. The cardiac impulse was heaving and diffuse, but the point of maximal impact was three and a half inches from the middle line in the fifth left intercostal space. A course thrill was felt along the carotids. A loud systolic murmur was present all over the precordial area, and most pronounced in the pulmonary region. The pulmonary second sound was accentuated. X-ray examination showed the heart to be considerably increased in size, chiefly as a result of left ventricular enlargement. The pulmonary artery was prominently dilated. Blood studies and electrocardiography gave no contributory information. A diagnosis of coarctation of the aorta was entertained and treatment was expectant.

On August 9, 1944, the patient was readmitted to hospital. The physical signs were essentially the same as before, except that a definite though soft diastolic murmur was heard at the left sternal border at the level of the second and third intercostal spaces. The basal murmur now seemed continuous, harsh in systole and faint in diastole. It was not a to-and-fro murmur. A tentative diagnosis of patent *ductus arteriosus* was made and exploratory thoracotomy was recommended. This was undertaken by Dr. B. T. Edye; but dissection of the mediastinum was not proceeded with,

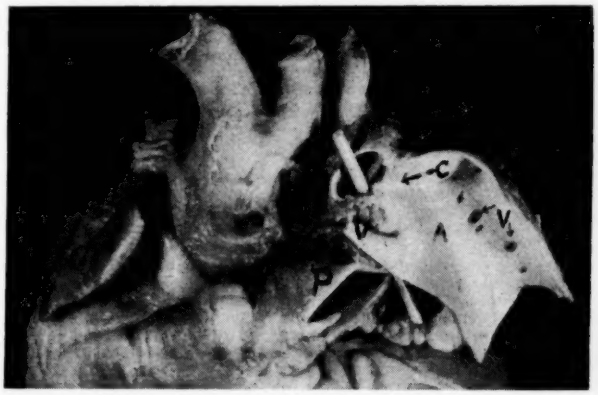

FIGURE V.

Case XIV. Photograph of heart with aorta opened and rotated anteriorly. A = aorta; P = pulmonary artery; V = vegetations; C = coarctation. The rod lies in the patent *ductus arteriosus*.

because the left subclavian artery was seen to be dilated, and no thrill was present at the roots of the great vessels as is usual in cases of patent ductus. Accordingly the thorax was closed. The child's recovery from the operation was somewhat slow, and although there were no complications, the episode seemed to upset her.

On April 21, 1945, the patient was readmitted to hospital on account of headache, dizziness, anorexia and feverishness with sweats. A blood count showed that the leucocytes numbered 20,000 per cubic millimetre. Culture from the blood identified the infection as pneumococcal. Despite treatment with full doses of sulphadiazine and penicillin (2,000,000 units per week), the patient died after fifteen days' treatment.

At autopsy the heart was enlarged (it weighed 240 grammes). Patent *ductus arteriosus* was present, and coarctation of the aorta was found at the point of entry of

the patent ductus (see Figure V). Vegetations were present at the orifice of the ductus, on the aortic cusps, on the left auricular wall and in the left ventricle. The aortic cusps were much thickened and rolled, and the valve was incompetent.

Although this patient was subjected to operation, the case was not one of uncomplicated patent *ductus arteriosus* and therefore is not included in an analytical discussion of signs in that condition.

Two further cases are worthy of mention, although the patients were not operated upon in this country and are not included in this series.

The first of these was a married stenographer, aged twenty-six years, who was first examined in March, 1944. Her history was that she had for long known that she had congenital heart disease. For two years she had suffered from infrequent attacks of palpitation lasting about half an hour. She had had dyspnœa on exertion for five years. She became easily fatigued. She had not, however, relinquished her work. She had been taking a daily ration of digitalis for eighteen months.

On examination, the patient was a normally developed woman. The systolic blood pressure was 170 millimetres of mercury and the diastolic pressure 60. Pistol-shot sounds could be heard along the peripheral arteries. A fairly well-marked funnel chest was found to be present. The apex beat was 13·5 centimetres from the middle line in the fifth left intercostal space. A thrill could be felt along the carotid arteries, but not elsewhere. A systolic murmur was present at the mitral area. At the base of the heart a harsh murmur was present throughout systole, and was prolonged beyond the second sound, fading, but not quite disappearing, in later diastole. It was most intense at the third left intercostal space at the border of the sternum. An electrocardiogram showed pronounced left axis deviation. X-ray examination revealed gross left ventricular hypertrophy with pulmonary artery dilatation. The red cells numbered 3,800,000 per cubic millimetre, the hæmoglobin value was 77% and the leucocytes numbered 10,000 per cubic millimetre.

This patient was referred to Dr. Paul White, of Boston, where she was operated upon by Dr. R. E. Gross.[16] During the operation, after the ductus had been divided, the heart ceased beating. With the aid of adrenaline, digitalis and manual stimulation the heart commenced to beat again. After some minutes, however, it again ceased, and all efforts at resuscitation were unavailing. Post-mortem examination failed to reveal any definite cause for this happening.

This case is mentioned on account of its similarity to Case XI above.

The other patient was a girl, aged three and a half years. She was very energetic and did not become fatigued easily, but if she ran hard she grew distressed and dyspnœic with a slight expiratory grunt. She had frequent respiratory infections.

On examination of the patient, speech retardation was observed. She could not make sentences and her speech was unintelligible. She was somewhat diminutive, her weight being only 28 pounds. The systolic blood pressure was 90 millimetres of mercury and the diastolic pressure 40. Systolic sounds were present over the femoral arteries. A systolic thrill was present at the base of the heart to the left of the sternum. The apex beat was in the fourth left intercostal space, five centimetres from the middle line. A typical continuous murmur was present in the second left intercostal space. The patient was referred to Dr. Gross, who operated successfully, dividing the ductus and oversewing the cut ends.

The case is mentioned here because of the speech retardation, similar to that observed in Case IX of this series.

Discussion.

From the point of view of diagnosis there is not one clinical sign which alone can be depended upon as being constant.

The typical machinery murmur may not always be found. In two cases the murmur at the initial examination was strictly limited to systole. The diastolic component developed during a period of observation of six months in one case and three years in the other.

X-ray pictures are invaluable in diagnosis, but are not pathognomonic. Dilatation of the pulmonary artery was present in only two-thirds of the cases. Left ventricular hypertrophy was found radiologically in all the foregoing cases, although in some it was slight. It must be borne in mind, however, that these were patients selected for operation.

Axis deviation occurred in the electrocardiograms in four cases and significant changes in the *S-T* intervals in two.

A clinical sign not described hitherto is pallor. It was noted in three-fourths of the cases and was found not to be related to anæmia. Although the latter was present in a mild form in some cases, it was insufficient to be of significance. A group of clinical signs common to aortic regurgitation and patent *ductus arteriosus* are enlarged left ventricle, raised pulse pressure, low diastolic pressure, visible arterial pulsation in the neck and pistol-shot arterial sounds. To these pallor may be added.

A systolic thrill was present in 11 of the 13 cases. In one case it was continuous and in another there was no thrill at all.

The blood pressure readings showed an increased pulse pressure in every case. In four cases a diastolic figure above zero could not be properly determined, the systolic arterial sound being sustained when the cuff was completely deflated. These were among the more serious cases. The effect of ligation was to reduce the pulse pressure in some cases considerably (for example, Case VII, 180 to 135 millimetres of mercury systolic and 100 to 95 diastolic). The systolic blood pressure also was reduced except in Case II, in which it was 10 millimetres higher.

The sex incidence in this series was predominantly among females, a finding which conforms to the reports of other authors.

Anginal pain on effort was described by two patients, both of whom were adults (Cases VII and XI). This is an unusual symptom in patent *ductus arteriosus*, and conversely, patent *ductus arteriosus* is an infrequent cause of *angina pectoris*.

The operative mortality rate was high as compared with figures reported from America. In two cases death was due to hæmorrhage, in one from the ductus itself, and in the other from the junction of the ductus with the aorta. These two operations were performed by different surgeons. The left pulmonary artery was found to be occluded in one case, and this may have had some bearing on the outcome. In another case death occurred after the operation had been satisfactorily concluded. This misfortune was similar to that which overtook the previously mentioned patient operated upon by Gross. Autopsy in this case failed to reveal any demonstrable cause of death. The occurrence of cardiac stand-still of this nature remains unexplained. It must be an infrequent event, as its description has not been met with in the literature. It would appear that the sudden closure of the ductus brings about an increase in peripheral resistance, to which a myocardium, suffering from long-standing strain, is unable to adjust itself at once.

No history of prenatal maternal rubella was obtained from any of these patients, this contrasting with the findings of other observers in Sydney.

The patients were carefully selected for operation on the basis of the above-mentioned indications. It was considered that a conservative palliative attitude had little to offer them as regards prognosis. Much experience has been gained, from both the medical and surgical points of view, and it is anticipated that much more favourable operative results will be obtained in future cases than in the early ones, as has been the experience elsewhere.

The results for the patients successfully operated upon have been uniformly good. Subjectively and objectively there has been a vast improvement in their well-being. All symptoms have disappeared and exercise and exertion are unaccompanied by any untoward effects. Some have indulged successfully in strenuous sport, such as competitive running and swimming. All the patients gained in weight except one adult, who was well beyond the period of growth. In Case VI the weight increased from 24·5 pounds to 40 pounds in a period of ten months. The thrill

TABLE I.

Summary of Results. Thirteen Patients Surgically Treated.

Case Number	Sex of Patient.	Age. (Years.)	Thrill.	Pallor.	X-Ray Findings.		Electrocardiographic Findings.	Blood Pressure. (Millimetres of Mercury.)		Anæmia.	Pounds' Weight Increase.	Period. (Months.)	Result.
					Dilated Pulmonary Artery.	Heart Smaller After Operation.		Before Operation.	After Operation.				
I	F.	6	+[1]	+	+	−[1]	O[1]	100/50	75/30	−	12	18	Well.
II	F.	8	+	+	+	+	O	105/0	115/55	+	13	15	Well.
III	F.	10	+	−	−	+	O	105/50	95/45	−	15	10	Well.
IV	M.	4	+	+	+		O	100/0		+			Died.
V	F.	20	+	+	+		Left axis deviation.	120/65		−			Died.
VI	F.	3	+	−	−	+	Abnormal *S-T* intervals.	95/0	80/45	−	12	7	Well.
VII	F.	35	+	+	+	+	Sagging *S-T* intervals.	180/100	135/95	−	0	7	Well.
VIII	F.	10	−	+	+	+	O	130/60	115/65	−	6	6	Well.
IX	F.	3	+	+	+	+	Exaggerated *T* waves.	70/0	70/35	+	5	5	Well.
X	F.	8	+	+	−		Left axis deviation.	110/55		+			Died.
XI	F.	24	+		+		Right axis deviation.	145/65		−			Died.
XII	M.	4	+	+	−		O	110/45	70/25	−	1	2	Well.
XIII	F.	14	+	+	O		Left axis deviation.	130/44	135/105	−			Well.

[1] "O" = normal; "+" = present; "−" = absent.

and murmur were abolished in every instance. The blood pressure readings returned practically to normal. The heart became smaller radiologically in all cases except one. The pulmonary artery shadow, when present, failed to disappear after ligation, but was generally smaller.

There can be no question as to the value of the operative treatment of patent *ductus arteriosus*. It has long since been carried beyond the experimental stage and has now been performed so frequently and with such satisfactory results as to have become a firmly established therapeutic procedure.

Summary.

1. The subject of patent *ductus arteriosus* is reviewed in a general way and 14 cases in which operation was performed have been recorded.

2. Four patients succumbed to operation, two from hæmorrhage and two from other causes. One died from subsequent infective endocarditis.

3. The patients successfully operated upon have been followed up for periods varying from two to twenty-one months, and the results are uniformly good.

4. Indications for operation are outlined.

5. The diagnosis is usually easy, but may be difficult.

6. The presence of cyanosis or clubbing practically excludes uncomplicated patent *ductus arteriosus*.

7. The prognosis in most cases is poor, apart from surgical treatment.

Acknowledgements.

I record with grateful acknowledgement my indebtedness to Dr. B. T. Edye, who operated upon most of the patients in this series, including the first two; also to Dr. M. P. Susman, who performed some of the operations. My thanks are also due to Dr. Clive Paton and Dr. S. V. Marshall for conducting the anæsthesia, and to the resident medical staff of the hospital for their work in the wards and operating theatre.

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SURGICAL TREATMENT OF PATENT DUCTUS ARTERIOSUS.

By B. T. EDYE,
Sydney.

I HAVE been invited by Dr. Bruce Shallard to contribute some remarks on our experiences of the surgical treatment of patent *ductus arteriosus*. I have operated upon ten patients, one male and nine females, whose ages ranged from three years and three months to thirty-five years. There were two deaths. One was due to hæmorrhage in an otherwise healthy girl, aged eight years, and the other to cardiac failure near the completion of the operation in a woman, aged twenty-four years, who had been suffering from precordial pain and other signs of distress on exertion. The anterior approach was used throughout the series, and the technique described by Dr. Robert E. Gross was followed, except in a few minor details. Briefly our procedure was as follows.

The patient was well prepared and was kept in hospital for several days or even weeks until the general health was rendered as satisfactory as possible. During this time the anæsthetist observed the patient from time to time and formulated the type of anæsthesia to be used. Immediately the patient was taken to the operating theatre, blood transfusion was established. As the skiagrams in all instances showed the position of the pulmonary artery to be opposite the second left intercostal space, an approach via this space was planned. An oblique incision over the intercostal space was employed. It coursed upwards and outwards in the direction of the fibres of the *pectoralis major* muscle from the margin of the sternum to the line of the anterior axillary fold. The muscle was split and not divided. In the older patients a curved incision was made with the convexity upwards to avoid the mammary tissue. The pleural cavity was entered by incision of the intercostal muscles and pleura, and the cartilages of the second and third ribs were divided. The internal mammary artery was sometimes divided between ligatures, but at other times the incision stopped short of it. If it was injured, it caused troublesome hæmorrhage and delay. A rib spreader was inserted over gauze sponges and the wound was widely opened. A light gauze pack was inserted to protect the collapsed lung. The diagnosis was confirmed by the characteristic thrill detected on palpation at or near the site of the ductus. Usually the anatomical structures, including the phrenic and vagus nerves and the left subclavian artery, were clearly visible. The ductus joins the aortic arch at or just beyond the level of the origin of the latter. This preliminary examination completed, the mediastinal pleura was divided vertically between the two nerves. The posterior leaf was raised to bring into view the recurrent nerve which hooks around the aorta immediately to the left of the ductus and is a most important guide to the latter. The ductus was also located by making pressure over the interval between the aortic arch and the pulmonary artery at varying points until the thrill was obliterated. The dissection of the ductus was a tedious process, fraught with considerable anxiety, for its wall was apt to be delicate and easily torn. The interval between the aortic arch and pulmonary artery was gently widened by blunt dissection with long curved artery forceps a little distance to the right of the ductus, and the process was repeated closer to the ductus until that aspect was freed. A similar dissection was carried out on the opposite side, the left recurrent laryngeal nerve being used as a guide. The forceps was then insinuated posteriorly so as gradually to open the interval between the ductus and the left bronchus behind. It was during this stage of the dissection that the ductus was apt to be torn. The manœuvre was assisted by counter-pressure with the left index finger in the interval between the aortic arch and the pulmonary artery, to the right of the ductus. Narrow malleable copper retractors were a useful aid in the exposure of the ductus.

The next step was the passage of a MacCormick dissector behind the ductus, aided as before by counter-pressure by the left index finger; care had to be taken not to catch the posterior aspect of the ductus with the instrument. When the dissector had been passed, no attempt as a rule was made to free the ductus any further posteriorly, but the dissector, now loaded with number 8 proofed silk, was passed and the ligature was drawn through. The ligature slipped through with greater ease when the dissector was *in situ*. A second ligature was passed. At or about this stage the ductus was compressed digitally and the effect on the circulation was noted. If after one or two minutes there were no untoward effects, the ligatures were carefully but firmly tied. By palpation and by auscultation with a sterilized stethoscope it was determined that the thrill and murmur had disappeared. The incision in the mediastinal pleura was sutured as well as possible. Any blood was aspirated from the pleural cavity and the chest wall was closed in layers. The divided costal cartilages were sometimes sutured with catgut and sometimes not. The ribs were drawn together with sharp hooks during the closure of the pleural cavity, and the lung was encouraged to expand by the anæsthetist and also by suction of air from the pleural cavity before final closure. No drainage was provided; convalescence was usually uneventful, and on occasions only was it necessary to aspirate pleural fluid. The blood transfusion was continued slowly throughout the operation.

Provided that the patients were in good general health and the anæsthetics well administered, the principal danger of the operation was hæmorrhage from a torn ductus. This alarming mishap was encountered twice. It occurred during the first operation as a result of the drag of a ligature during its passage behind the ductus. Fortunately it was controlled immediately the ligatures were tied. In the second instance a ligature towards the aortic end was in the process of being tied when it cut through the ductus flush with the aortic wall. In spite of the rapid transfusion of several litres of blood and various attempts to repair the defect in the aorta, the patient succumbed. It was because of the fear of hæmorrhage that I considered it important to have the transfusion established before embarking on the operation proper. The internal saphenous vein at the ankle was selected to keep those in charge of the transfusion away from the field of operation. The blood was allowed to drip slowly, but in case of emergency the rate was increased at once, while hæmorrhage was checked by digital or other pressure and plans were made for its permanent control.

It is possible that when the cardiac efficiency has been impaired, the introduction of a large amount of blood may be detrimental. Whether this was in any way a contributory cause of death of the adult patient who died towards the completion of the operation is a moot question.

Acknowledgements.

The operation for ligation of the patent *ductus arteriosus* is one in which efficient team work is necessary for success. This satisfactory state was attained at the Royal North Shore Hospital of Sydney, where the operations were performed. The selection of the patients was supervised by Dr. Bruce Shallard, and all operations were undertaken with the cooperation of Dr. M. P. Susman. The anæsthetics were administered by Dr. C. N. Paton and Dr. S. V. Marshall, and the blood transfusions by the resident medical officers, who also were ready to help in other directions. The operating theatre staff were as enthusiastic and efficient as could be desired.

ANÆSTHESIA IN CASES OF LIGATION OF PATENT DUCTUS ARTERIOSUS.

By CLIVE N. PATON,
Sydney.

IN the series of patients with patent *ductus arteriosus* operated upon by Dr. B. T. Edye at the Royal North Shore Hospital of Sydney substantially the same form of anæsthesia was used in all cases. This consisted of basal narcosis established with a basal dose of "Avertin", followed by inhalation anæsthesia with cyclopropane.

The majority of the patients were young children, and basal narcosis was first resorted to in these cases, in order to secure a quiet approach to anæsthesia and a rapid and uninterrupted induction. It achieved this object so successfully that it became the routine method in all cases. It has the additional advantage of securing slow, shallow respiration (essential to the performance of the surgical task) and a satisfactory period of narcosis after operation.

Cyclopropane was selected because of its non-toxic and non-irritating features, the shallow type of respiration it produces, the ease with which "controlled respiration" can be instituted, the high concentration of oxygen in which it is administered, and its reversibility.

A standard McKesson machine was used in all cases; this facilitated the application of accurately measured positive pressure.

Premedication.

To the adult patients morphine sulphate (one-sixth of a grain) was given one and a half hours before operation. A full basal dose of "Avertin" was given half an hour later, and atropine sulphate (1/100 grain) was given half an hour later still. In the case of children the morphine was omitted and the dose of atropine sulphate was reduced to 1/150 grain.

Procedure.

On the patient's arrival at the operating theatre, venoclysis was effected in the malleolar vein of one or other side. A Hamilton-Bailey cannula was used to commence slow transfusion of stored citrated blood. (Typing and cross-matching were carried out in advance as a routine measure.) A slow rate of drip (15 to 20 per minute) was established.

The naso-pharynx, pharynx and larynx were next lightly sprayed with a 5% solution of cocaine hydrochloride prior to induction of anæsthesia. This was done to facilitate intubation and to forestall any tendency to "gagging" or laryngospasm.

Induction was carried out with cyclopropane along the usual lines. In adult patients transoral intubation was carried out by means of a number 6 Magill tube fitted with a Waters inflatable cuff. In the case of the youngest children intubation was not carried out, and in the older children transnasal intubation was carried out, the pharynx being packed with paraffin-impregnated gauze. Carbon dioxide absorption was then introduced.

Whilst it is realized that intubation is not essential, it has the advantages of providing an infallible airway, of reducing dead space in which carbon dioxide may accumulate, and of facilitating the introduction of "controlled respiration" if required, or of artificial respiration or positive pressure.

"Controlled respiration" was instituted when possible during the more exacting surgical manœuvres. However, respiration was generally quiet and shallow and anæsthesia uneventful.

The smaller children, contrary to general opinion, appeared to suffer no disadvantage from the use of the large closed circuit apparatus.

No great depth of anæsthesia was required for the most part, a moderate second plane level being sufficient. Anæsthesia was deepened as a rule as the pleura was opened.

Slight positive pressure was applied at intervals of fifteen minutes during the operation in order partially to expand the collapsed lung. This procedure was carried out again during closure of the chest wall, to assist in reexpansion of the collapsed lung. At this stage the anæsthetic mixture was changed to nitrous oxide (80%) and oxygen (20%). Carbon dioxide absorption was discontinued to allow of five minutes' overbreathing at the conclusion of the operation, as a safeguard against atelectasis.

Complications.

Complications either during or after operation were surprisingly few.

Tachycardia occurred in three cases. In one child it occurred during induction but disappeared as anæsthesia progressed. In another child, aged fourteen years, tachycardia resulted each time an attempt was made to deepen the plane of anæsthesia. A change was made to nitrous oxide and oxygen with ether, and the tachycardia disappeared.

The third patient, aged twenty-three years, was one of the subjects who succumbed during operation. Soon after commencement of the operation she exhibited tachycardia and tachypnœa. Both became progressively worse. "Digoxin" was twice administered intravenously without avail. After ligation of the ductus she exhibited progressive cyanosis with a pulse rate reaching as high as 150 per minute. The chest was closed rapidly and after a few minutes respiration and cardiac activity ceased.

Cardiac arrhythmia was later sometimes noted when anæsthesia was deepened. This disappeared on return to a lighter plane; apparently it consisted of simple auricular extrasystoles.

Hæmorrhage occurred in two cases. In the first (the first case in which operation was undertaken) the circulation was adequately supported by opening up of the transfusion rate to a maximum until surgical control of the hæmorrhage was effected. In the second (the patient was a child aged nine years) the hæmorrhage was devastating. A second transfusion was installed in the opposite leg, and by running both at a maximum rate, five litres of blood were given in one and a half hours. The hæmorrhage, however, proved uncontrollable. Transfusion was discontinued in each case as soon as the ductus had been successfully ligated.

Recordings of blood pressure and of pulse and respiration rates were kept in every case.

The dramatic rise in diastolic pressure, from its customary low level in these cases to 80 to 90 millimetres of mercury after ligation, was noted consistently.

Auscultation of the exposed heart was carried out on several occasions, immediately after ligation, and the prompt disappearance of the gross preoperative bruit was noted.

Conclusions.

1. Basal narcosis with "Avertin", followed by inhalation anæsthesia with cyclopropane, was found a satisfactory from of anæsthesia in a series of cases of ligation of a patent *ductus arteriosus*.

2. Endotracheal intubation is an added advantage for the reasons set out.

3. A minimum of disturbance of cardiac and respiratory function was experienced.

HYPERSENSITIVITY TO HORSE SERUM.

By P. WARD FARMER,

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REACTIONS to horse serum occur not infrequently, and may be serious, even causing death, or simply mildly inconvenient to the patient. Antitoxin is nearly always prepared from horse serum, and while immunization and treatment with antitoxin are carried out, an understanding of the "allergic" reactions that can occur after serum administration is extremely important.

Pirquet and Schick[1] first clearly described serum disease in 1905, and they observed that when second injections of horse serum were given after an interval of four months or more, symptoms of serum sickness were accelerated; also that if a second injection was given in less than four months, the reaction might be accelerated, or it might occur immediately within minutes or a few hours.

Types of Serum Reaction.

Vaughan[2] describes four types of reaction.

1. *Delayed Reaction or Serum Sickness.*—A person who is not allergic to horse serum may, after a first injection, become so after an interval of about ten days. Horse serum continues to circulate in the body until antibodies appear. This may be regarded as evidence of beginning sensitization, and the symptoms of serum sickness occur because the antigen is still present.

2. *Accelerated Reaction.*—An accelerated reaction follows reinjection of serum after a longer interval than four months. Very early reactions may be similar to the immediate type, or delayed reactions may be indistinguishable from serum sickness.

3. *Immediate Reaction.*—An immediate reaction may follow reinjection of serum given after the incubation period of ten days or up to a period of about four months, and resembles a constitutional reaction as observed after an overdose of some other allergen, as for example pollen extract.

4. *Natural Serum Sensitivity.*—The reaction resulting from natural sensitivity to serum occurs in a person naturally sensitive to horse serum or horse dander. Desensitization is much more difficult than in the "artificially sensitized" person, owing possibly to the higher degree of sensitivity or to the manner of reaction to the atopen. The symptoms are similar to those of the immediate type of reaction. Most really serious reactions seem to occur from first injections of serum.

Symptomatology.

Ordinary Serum Sickness.—In ordinary serum sickness there is an incubation period, owing to the time necessary for the formation of antibodies. Skin tests usually give negative results, but later may yield positive results. The reaction usually occurs seven to twelve days after the administration of the serum, but the interval may be more or less. The characteristic symptoms are fever; an eruption, which may be urticarial, morbilliform or scarlatiniform; less commonly, adenopathy, arthritis, albuminuria, nausea, vomiting; and rarely, nervous manifestations such as meningeal irritation, bulbar palsy or neuritis. Rash and fever are the most constant symptoms. A recurrent form of serum sickness may be encountered.

Accelerated Serum Disease.—If a previous serum injection has been given, the "accelerated" type of reaction may occur after the next injection. The symptoms are similar to those of serum sickness, but come on after a shorter incubation period (up to four days); this type of case can be anticipated by the demonstration of positive results to skin tests with horse serum. The symptoms are usually more alarming than those of ordinary serum sickness.

Allergic Shock.—In the case of allergic shock, the subject is often naturally sensitive to horse serum, and frequently sensitive to horse dander. Tuft[3] has shown that horse serum contains two antigens, dander antigen and serum antigen, and the patient may be sensitive to one or both. Previous injections of serum will also highly sensitize the patient to yield this reaction. The reaction may occur at once or be delayed up to an hour, but rarely more. The more intense the reaction, the more it resembles general shock. Dyspnœa is constant without asthmatic breathing, and pulmonary œdema is the characteristic response; there is a greater probability of recovery if wheezing, sneezing or urticaria develops. The necropsy findings are similar to those found in experimental anaphylaxis; in some subjects hepatic and splanchnic engorgement is present, in others, distension and emphysema of the lungs. Failure of the right side of the heart is frequently present, as in the rabbit in a state of anaphylactic shock. These subjects may fail to respond to adrenaline, and the blood pressure drops quickly.

Thermal Reactions.—Although thermal reactions are not a true anaphylactic response, they are a complication of serum therapy. They occur not infrequently and may be severe enough to cause death. The reaction usually occurs fifteen to seventy minutes after the injection of serum, and is mostly preceded by a chill. For diagnosis it is safer to take the temperature every half hour, or more often, if chill, restlessness or delirium occurs, or if the patient complains of feeling hot. The temperature may reach 107° to 110° F.

Factors in the Acquisition of Serum Allergy.

The patient may become reactive from a first injection of serum, and repeated injections are more likely to produce sensitivity. Kojis[4] found that after repeated intramuscular injections of diphtheria antitoxin there is a 50% greater incidence of serum sickness than after a primary inoculation, while anaphylactic shock is fourteen times more frequent. A patient seems more likely to become sensitive when there is a family history of allergy. The site of a previous injection has an influence, and the intravenous route appears most likely to produce sensitivity. A large quantity of serum injected seems to produce sensitivity more frequently than a small quantity. Age is a factor, as children seem to develop serum sickness more readily than adults.

Mortality Rate.

Park[5] held that the mortality rate from serum anaphylaxis was only one in 50,000 cases, whereas Kojis[6] had one death in every 1,242 cases. It is probable that figures quoted by earlier authors are misleading.

Hyperthermia accounted for six deaths in a series of 6,211 cases reviewed by Kojis.[7] This type of reaction may occur after any other infusion, and is not peculiar to serum administration.

Prophylaxis.

Prophylactic injections of serum should not be given except when clearly indicated. People who run a risk of developing tetanus owing to their occupation should be immunized with tetanus toxoid; wholesale immunization with diphtheria toxoid should reduce diphtheria to a minimum. Reinjection of horse serum should be avoided as far as possible, especially within three or four months after the first injection of serum. A dangerous period for reinjection appears to be fifteen to twenty days after a previous injection. Intravenous injections should be avoided. A history of any kind of allergy in the patient, especially sensitivity to horse dander, is extremely important, and a family history of allergy would make one more cautious still. A negative reply to questions on this matter cannot be regarded as an adequate safeguard. I have found[8] that many subjects of juvenile eczema are sensitive to horse dander and so may be allergic to horse serum. Skin testing with horse serum should be performed in every case in which there is a history of allergy, especially to horse dander, or in which a previous injection of serum has been given.

Skin Testing.

A safe procedure for skin testing is as follows. It is better to use normal horse serum than therapeutic serum. Rubber-capped bottles containing sterile diluted horse serum can be kept at hand, and for intradermal testing needles should be sharp, but should have a short bevel. In case of a reaction, have ready a solution of 1/1,000 adrenaline chloride solution and also a tourniquet. It cannot be stressed too strongly that it is dangerous to commence an intradermal test with undiluted serum. Inject intradermally into the volar aspect of the arm 0·01 mil of a 1/100 solution of horse serum diluted in normal saline solution; about half-way between the shoulder and elbow-joint is a convenient place. A control test of 0·03 mil of sterile normal saline solution is injected intradermally about three inches below the previous injection, and the result is watched for twenty minutes. If there is no weal formation, erythema or itching at the site of injection, the result can be regarded as negative. The test is then repeated with a one in ten dilution of serum, and if this result is negative it is safe to proceed with the injection of undiluted antitoxin. Coca[9] states that if there is a reaction to the 1/100 dilution, and if the patient is known to be sensitive to horse dander, the injection of serum is

contraindicated. In this type of case desensitization will probably fail. If, however, an intradermal test of a one in ten solution gives negative results in a subject sensitive to horse dander, cautious hypodermic administration may be proceeded with. When it is vitally necessary to give antitoxin and the patient reacts to a one in ten dilution of serum, desensitization may be attempted.

Desensitization.

Make each injection into different localities in the same arm, and inject deeply, as some painful local swelling may occur. Make certain that the point of the needle is not in a vein. It is wise to wait twenty minutes between each injection to see if a constitutional reaction occurs. Inject successively 0·1 mil, 0·2 mil, 0·4 mil, 0·6 mil and 0·8 mil of the one in ten dilution of serum. If no general reaction occurs between each injection, repeat the same procedure with undiluted serum, till all is given. If a constitutional reaction should develop, apply a tourniquet above the site of injection and inject 0·5 mil of a 1/1,000 adrenaline chloride solution above the site controlled by the tourniquet. No more serum should be injected until the tourniquet has been released for forty to sixty minutes without further recurrence of anaphylactic symptoms. The next dose should only be 30% of the preceding dose, and subsequent doses should not be increased by more than 50%. For rapid desensitization it is wise if possible to have the assistance of a trained allergist.

Treatment.

Serum Sickness.—In serum sickness the itching is usually controlled with calamine lotion containing phenol (1%) or by a warm soda bath (one cupful in a bath). If these measures fail to give relief, ephedrine sulphate (0·5 grain orally) or a subcutaneous injection of adrenaline will help. Aspirin, phenobarbital or even morphine may be necessary. For nausea and vomiting, sips of a solution of sodium bicarbonate should give relief.

Allergic Shock.—In allergic shock, inject immediately 0·5 mil of a 1/1,000 solution of adrenaline chloride. If no response is obtained with this dose, larger doses seem valueless. If there is no response to adrenaline the outlook is grave. It is important to remember that an overdose of adrenaline may be dangerous. A tourniquet should be applied above the site of injection of serum and released at intervals of ten minutes. Shock may be combated by injections of morphine sulphate and by the application of heat to the body. Hot drinks may be given when the patient can take them.

Thermal Reaction.—It is important to know what treatment should be instituted for a thermal reaction, as it has to be prompt and heroic. Colonic irrigations of warm water should be given immediately, and there appears less chance of recovery if the treatment is given more than one hour after the onset of hyperpyrexia. Salicylates and ice bags do not appear to help this condition.

Some Illustrative Cases.

CASE I.—A female patient, aged twenty-one years, was referred to me from the country, because of severe allergic shock occurring after a prophylactic injection of tetanus antitoxin. She had never had a previous injection of serum. Dyspnœa and cyanosis developed immediately after the injection. She recovered after several injections of adrenaline chloride. Urticaria was apparent two hours later, and five days later again it reappeared. Skin tests produced a large reaction to horse serum, but no reaction to horse dander. I attempted desensitization, starting with a 1/1,000 solution of horse serum, but could not reach an undiluted strength, as the reactions after injections were so severe. There was a strong family history of allergy, and the patient appeared to be one of the naturally sensitive people. It would be extremely dangerous for her to have another injection of serum.

CASE II.—A female patient, aged twenty-seven years, had severe anaphylactic shock after being given tetanus antitoxin; she reacted immediately and lost consciousness in a few seconds, while dyspnœa and cyanosis were observed. She was revived with injections of adrenaline chloride. She had had no previous injection of serum. She suffered from mild attacks of asthma and knew that horses "upset" her. Scratch tests produced large reactions to horse serum and horse dander.

CASE III.—A male patient, aged eight years, was referred to me because of intermittent urticaria and pruritus following a tetanus antitoxin injection given three weeks previously. He had had a previous injection of tetanus antitoxin three years before without any untoward symptoms. Skin tests, including tests with horse serum and horse dander, produced negative results.

CASE IV.—A female patient, aged fourteen years, was given tetanus antitoxin following an injury. Allergic shock occurred in ten minutes. I saw the child one hour after the injection had been given; her condition was serious; she had pronounced dyspnœa, and the pulse at the wrist was almost imperceptible. She had been given adrenaline promptly, and two hours later a wheeze could be heard in the chest. She made a good recovery, except for the occurrence of urticaria on the fifth day. She suffered from perennial hay fever, and skin tests produced positive reactions to horse dander and horse serum.

This patient appears to be naturally sensitive, as there was no history of a previous injection.

CASE V.—A male, aged twenty-seven years, had an injection of tetanus antitoxin following an injury. Intense giant urticaria developed in two hours. The urticarial condition disappeared in three hours. Four years previously he had had a prophylactic injection of diphtheria antitoxin without untoward symptoms. Skin tests produced large reactions to grass pollens; otherwise the results were negative. The patient suffered from seasonal hay fever.

Comment.

These cases are typical of those I have encountered in practice. In Cases I, II and IV the condition resembles in all respects the acute anaphylactic shock observed after an overdose of some other allergen such as pollen extract, and the response to adrenaline was similar. The patients appeared to be of the "naturally sensitive" type. In Cases III and V the reaction would appear to be of the "accelerated" type.

In most cases a severe reaction develops very soon after the injection is given. Severe reactions and fatalities are caused by failure to realize the harmful potentialities of a serum injection, by omission of skin tests in the case of a possibly sensitive subject, and by too rapid desensitization. In the case of natural sensitivity, the dangerous period appears to be soon after the administration of the first few mils.

Conclusion.

The types of reaction that may follow injections of horse serum are summarized, and the relation between horse serum and horse dander sensitivity is stressed. These reactions appear to be similar in type to those following an overdose of any other allergen injected. The detection of sensitivity to horse serum by skin testing, and a method of desensitization, are suggested. The "naturally sensitive" person appears most likely to develop dangerous symptoms.

Acknowledgement.

I wish to thank Dr. Charles Sutherland for helpful advice and criticism.

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A REPORT OF THE TREATMENT OF SEVEN CASES OF GRANULOMA VENEREUM WITH PENICILLIN.

By F. BOYD TURNER,
Major, Australian Army Medical Corps.

THE problem of *granuloma venereum* was discussed with Professor Sir Howard Florey during his visit to the Northern Territory in 1944, particularly in relation to those cases in which the condition progresses and does not respond to injections of the various antimony preparations, and in which an extensive ulcerated area is produced. As a result of these discussions it was decided to try the effect of penicillin, at first parenterally and then locally, in eight cases of *granuloma venereum* at the Native Hospital, Tennant Creek. To enable this to be done, the patients were transferred to an Australian general hospital where adequate facilities were available.

History and Condition of Patients on Admission to Hospital.

One patient had been admitted to hospital in March, 1944, but all the other patients had been in hospital over twelve months. All except one had had extensive ulceration before admission to hospital, and all since admission to hospital had had several series of antimony injections, of "Fouadin", "Anthiomalin" and "Neostam", and several had been given courses of "Novarsenobillon" and "Myocrisin".

In all cases except one extensive ulceration of both labia was present, extending in some cases to the thighs and perineum, and to the sacrum in one case. The inguinal and femoral lymph glands were enlarged in all cases, and in one case had broken down, with the production of a chronic ulcer. All patients had an elevated temperature, swinging between 99° and 102° F., and all had a rapid pulse rate and general signs of toxæmia. They were unable to move comfortably and their appetite was poor.

In one case the ulcerated area had a rolled-up, firm edge, and proved to be a carcinoma.

Pathological Findings.

In all cases except one similar changes were found in both smears and microscopic sections, and cultural findings were similar.

In smears from the ulcers many pus cells, a few mononuclear cells and an occasional large form measuring approximately 30μ in diameter and containing indefinite intracytoplasmic bodies were found. The intracellular bodies were much smaller than a coccus. The nucleus occupied more than half of the large cells. Plasma cells were present, and in some smears they were the predominant cells. In all the smears numerous organisms were found, including cocci, *Bacillus coli communis*, fusiform bacilli and a spirochæte in one case.

The following organisms were grown on culture in one or more cases: Friedländer's bacillus, hæmolytic and non-hæmolytic streptococci, *Bacillus subtilis*, *Bacillus coli communis* and *Staphylococcus aureus*. One or more of the organisms were isolated in each case.

In all sections from the ulcers the plasma cell predominated; chronic inflammatory changes were conspicuous. Toward the surface suppuration was evident and was associated with loss of the epithelium. At the ulcer margin epithelial proliferation was found. Local accumulations of chronic inflammatory cells were seen immediately below the epithelial cells. Large mononuclear cells were seen in some sections.

Examination of sections from lymph nodes revealed mild inflammatory changes of a non-specific character.

Major T. Constance, who carried out the above investigations, commented as follows: "Although the findings are not conclusive, the microscopic appearances are consistent with the diagnosis of *granuloma venereum* with secondary infection."

The one exception was the case of Topsy K. Examination of sections revealed a squamous-cell carcinoma, and in the smear pus cells and epithelial cells, but no abnormal mononuclear cells were found.

Kline and Kahn tests were carried out in those cases in which there was no previous record that these tests had been done; four of these tests were performed with two positive and two negative results. Among the other cases (four, including the case of carcinoma) the test had produced negative results on two occasions in one case and positive results on several occasions in the other three. Of the seven granuloma cases the Kline test produced positive results in four and negative results in three.

Treatment and Progress.

On October 24, 1944, parenteral treatment with penicillin, 30,000 units every three hours during the day and every four hours at night, was commenced in three cases. On October 31 a similar course of treatment was started in the other four cases.

Parenteral penicillin treatment was discontinued in both series on November 13, and the local application of penicillin cream was started. The cream contained 500 units of penicillin per gramme. The patients in Group I received a total dosage of 4,110,000 units of penicillin parenterally, and those in Group II, 2,490,000 units.

The local treatment consisted of bathing the affected area twice a day and of the application of a sterile pad, until local penicillin therapy was substituted for parenteral administration.

Clinically all the patients showed great constitutional improvement; the temperature became normal or was only slightly elevated, the pulse rate fell correspondingly, their appetites improved, and they became generally more active. The ulcerated areas became cleaner, the discharge was less, the enlarged gland subsided, and the granulation tissue became flatter and level with or below the skin margin.

On November 10 smears were taken from all the patients, whilst sections were taken from one patient in each series. The smears presented the same cytological picture as before, including the large form of plasma cell and inclusion bodies. On the other hand, organisms were not numerous. The report on the microscopic examination of the sections was as follows: "The histological picture has not materially altered. The most striking feature is the presence of large nodular masses consisting of round cells. Plasma cells are conspicuous. Secondary infection is *still* evident but is not as marked as on previous examinations."

All the patients, after the cessation of parenteral administration of penicillin and the local application of penicillin, continued to show some improvement, but the ulcerated areas became moister. From November 23 sulphanilamide powder was dusted on the ulcer area and then the penicillin cream was applied. All patients continued to make progress, with the exception of one who died, and the ulcerated areas became less moist. On December 12 the penicillin cream and sulphanilamide applications were discontinued and *Unguentum Hydrargyri Oxidi Flavi* was substituted.

The individual progress of the patients was as follows:

Group I.

The patients in Group I received 4,110,000 units of penicillin parenterally.

One patient, "Corky", died on November 27 after a rise of temperature and pulse rate with abdominal discomfort and œdema of the abdominal wall and legs. At autopsy she was found to have a thin-walled, distended bladder and pyonephrosis. Examination of sections of the kidney revealed generalized overgrowth and interstitial fibrous tissue with considerable infiltration by polymorphonuclear leucocytes. There was a decrease in the number of glomeruli, and many tubules were degenerated. The cause of death was pyonephrosis and *granuloma venereum*.

"Sally" at first showed signs of rapid healing of her condition; but after reaching a stage at which the ulcerated area was about three-quarters of an inch wide, extending from the pubes around the right *labia majora* to the perineum, she has since shown no progress. Examination

of the sections of the ulcer margin taken on November 10 reveals strands of squamous epithelium growing down through the basement membrane, but there are no mitotic figures.

This is probably not a malignant change, but similar to the epithelial changes at the margin of areas of chronic cervical erosion *et cetera*.

"Nellie", the third member of Group I, maintained a steady improvement, and now three-quarters of the area has become epithelialized and shows every sign of healing completely.

Group II.

The patient in Group II received 2,490,000 units of penicillin parenterally.

One patient, "Janie", who had the most extensive ulceration extending from the pubes to the femoral triangles and the sacrum, died on January 20, 1945. The ulcerated arch had remained clean, and in isolated areas showed signs of healing. At autopsy advanced bilateral pulmonary tuberculosis was discovered.

"Topsy L." showed great improvement in her general condition, the granulations became flat and clean, and the edge showed signs of epithelial growth; but the other areas have extended, and at present some areas are healing and others breaking down.

"Dora" showed continued progress; now more than three-quarters of the area is epithelialized, and it appears certain that the whole area will be covered.

The condition of "Daisy", who had lymph vulva and not very extensive ulceration, was completely healed by December 8, 1944, and has remained healed.

Comment.

Thus of the seven patients, two have died, the lesion of one is completely healed, that of two is healing rapidly and the ulcerated area will almost certainly be completely epithelialized, the condition of one has improved considerably but now has reached a stationary condition, whilst the last has an active condition with healing in some areas and fresh ulceration in others.

Examination of smears taken on February 11, 1945, from all the patients with any ulcerated area revealed the typical intracellular bodies associated with granuloma, with the exception of smears from "Sally".

The Kline test, to which Sally and Topsy L. reacted when tested on October 18, 1944, still produced a positive result on December 18, 1944, and on February 24, 1945.

Discussion.

From the progress of the patients and the pathological findings it is evident that penicillin had no effect on the *granuloma venereum*, but did overcome the secondary invaders and allow epithelialization to occur. This will perhaps break down again if a further course of antimony is not given, the heavy secondary infection having previously prevented the antimony from having its specific effect.

The discovery in one case of squamous-cell carcinoma of the vulva among eight suspected cases of granuloma, and in another of a possible precancerous change in the epithelium, suggests the possibility that a cancerous change may be a complication of granuloma.

The complication of pyonephrosis and pyelonephritis is easily understood from the heavy secondary infection and fibrous tissue reaction of the ulcer base, which in this case involved the urethra.

Conclusions.

Penicillin has no direct effect on *granuloma venereum*, but will be valuable in clearing up the secondary infection and so allowing the specific treatment with antimony to be more effective.

The positive Kline reaction was unaltered fifteen weeks after parenteral treatment with large doses of penicillin had been discontinued.

Acknowledgements.

This investigation has been carried out by various members of the medical service. Major T. Constance reported on the first and second series of sections and smears, whilst Captain A. C. Christie, with Dr. W. I. B. Beveridge, of the Walter and Eliza Hall Institute of Research in Pathology and Medicine, reported on the last series. Captain L. E. Verco and Captain M. Main assisted in the clinical management of the patients. Lieutenant-Colonel Gwyn Williams kindly granted facilities for these investigations to be carried out at an Australian general hospital. The Director-General of Medical Services has given permission for the publication of this paper.

THE TREATMENT OF FURUNCULOSIS.

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WITHIN the last two weeks two patients came to be treated for furunculosis. One had been treated with manganese on the ground that "the old remedies are the best", and the other was told that his doctor "did not believe in treatment by injections". His boils were lanced. Both patients were worse after treatment. I know by experience that the only treatment is by a combination of such new-fangled remedies as the sulphonamide drugs or penicillin and injections of a vaccine.

The object of treatment of any infection is threefold: to cure the disease; to prevent immediate or remote recurrence in those infections, an attack of which does not confer immunity; and to prevent spread to others. The third factor is of enormous importance in furunculosis, since free pus on an open surface may endanger the lives of a long train of individuals distant from the original focus, secondary infection of surgical wounds being a special risk.

Furunculosis is an infection of hair follicles by *Staphylococcus aureus*. The infection is usually localized as a "boil", but spreads to other follicles in pus from a boil which ulcerates or is opened surgically. Treatment by manganese, tin *et cetera* is futile, while surgical treatment is illogical and dangerous. When a boil is opened, nothing can possibly prevent spread of infection to other follicles and to other individuals, while incision does nothing, of course, to confer immunity. A boil should never be opened.

I wish to publish a method of treatment which has almost never failed. Statistics are unnecessary, since they would simply record rapid, complete and usually permanent cures in well over 500 cases. Many of these patients had had rapidly recurring crops or continuous boils over many months, sometimes over years; one patient had had 140 without a break. The uniformly and immediately favourable response in every case cannot possibly be assigned to chance, since in at least a majority of cases the chances were distinctly in favour of persistence and certainly of recurrence.

The treatment consists of a combination of sulphonamide drugs and an autogenous vaccine. I have found penicillin necessary in only one case; this patient, who refused vaccine treatment, relapsed the day after his penicillin treatment stopped. Thus sulphonamide drugs and penicillin, while they undoubtedly clear up the immediate lesions, do nothing to prevent recurrence after treatment by them is suspended.

I have used sulphapyridine, sulphathiazole and sulphadiazine administered to saturation point in four days, and followed by one to two grammes per day until 25 grammes have been consumed. I have discarded sulphapyridine because of its occasionally nauseating properties, though its therapeutic efficiency is little if anything below that of the other two.

Vaccine Treatment.

Vaccine treatment is used to reinforce immediate cure and to confer immunity against recurrence. The preparation of the vaccine must be done with great care and a full knowledge of the relevant factors. I have found by experience that vaccines made from stock cultures, or sub-

cultures, or primary cultures after a certain time, are useless, as are vaccines of low dosage made from a very young primary culture.

It seems to me, speaking empirically, that the antigenic factor is a component of the earliest growth phase of the *Staphylococcus aureus*. My custom is to sow pus from a boil completely over a freshly poured agar slope, pH 7·2, in a test tube with an inside diameter of five-eighths of an inch. This is done late in the afternoon, and the vaccine is harvested not later than 9 a.m. next day. The culture, that is, should be about fifteen or sixteen hours old, and I have found that after eighteen hours the antigenic value of the vaccine diminishes rapidly. The dosage of vaccines formerly was altogether too timid. I suspend the whole of the agar slope in 10 mils of saline solution and sterilize it with a 0·5% solution of carbolic acid. To heat the vaccine is both unnecessary and harmful. This gives a vaccine with a dense opacity due to a number of organisms almost impossible to count; the strength would probably be of the order of 20,000,000,000 organisms per cubic millimetre. This dosage is tolerated without the slightest inconvenience beyond a soreness of the arm following the first injection.

Doses of 0·25 mil, 0·5 mil, and a stock dose of 1·0 mil are given at intervals of five days for six to eight doses. A rest of three weeks is given, and a "booster" dose of 1·0 mil is injected. Occasionally a second, and rarely a third, "booster" dose is given.

Comment.

This method of treatment will give complete and permanent cure of furunculosis in the vast majority of cases. A small minority of patients may require a complete repetition, but in the end they all yield to treatment.

Since neither vitamin *A* nor any other vitamin is in the slightest degree involved, diet need not be considered beyond the patient's being advised to consume a well-balanced diet for ordinary hygienic reasons.

Summary.

Furunculosis can be satisfactorily treated by a combination of autogenous vaccine prepared from a phase I culture, with sulphathiazole or sulphadiazine, or rarely in urgent cases, with penicillin.

AN EPIDEMIC OF DIARRHŒA CAUSED BY A NEW STRAIN OF THE SALMONELLA GROUP.

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AND

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WITH AN APPENDIX BY NANCY ATKINSON,
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IN the early spring of 1944 an epidemic of diarrhœa occurred in our hospital area. The onset was gradual. A member of the nursing staff was admitted to hospital suffering from vomiting, diarrhœa and fever. From her stool a Salmonella-like organism was isolated. This appeared to be an uncommon strain, and we were uncertain at this stage whether its inclusion in the Salmonella group was justifiable.

Two days later three further members of the hospital staff reported with similar symptoms, and on clinical grounds one of these appeared to be suffering from a Salmonella infection. Appropriate enrichment methods for the detection of Salmonella organisms were therefore instituted in the laboratory. Cases occurred thereafter almost daily for nine days in all, after which new cases ceased quite abruptly. Many cases were extremely mild, and some were detected only by direct questioning of contacts. A total of 29 cases was investigated in the nine-day period of the epidemic, and the same Salmonella-like organism was isolated in 26 of them.

Clinical Findings.

The salient clinical features are set out in Table I, covering the 26 patients from whom the organism was isolated.

Abdominal pain was a symptom in 22 cases and was accompanied by tenesmus in six cases. Vomiting was not troublesome. Those patients who were affected vomited only once or twice at the onset of their illness. Four patients complained of headache, one of thirst, and two of cramp in the legs. Dehydration did not develop in any instance. Only four soldiers were really ill, none were placed on the seriously ill list, and the mortality was nil.

Leucocyte counts performed on two patients revealed no abnormality. In one of the more severe cases at the height of the fever, attempted culture of organisms from the blood was unsuccessful. Sigmoidoscopy was performed at an early stage of the illness in a few cases; the appearances were normal, apart from mucosal congestion, which could be merely a result of hurried bowel action.

The common type of stool was fluid, and often contained mucus which was usually acellular or epithelial in nature. In eight instances pus cells and sometimes a few red blood cells were present, so these were classed as "indefinite exudate". In two cases the mucus was frankly blood stained and showed the cellular characters of a "typical bacillary exudate".

The cases may be divided roughly into three groups. First there was the mild type, in which the patient retired to bed feeling quite well and woke in the early hours of the morning with abdominal pain and diarrhœa. He passed three to ten or twelve stools in about twelve hours, after which the diarrhœa ceased and soon he felt quite well. Sometimes there was an initial rise in temperature, sometimes transient nausea and occasionally vomiting. Clinical examination revealed no abnormality, except perhaps a coated tongue. More than a third of the cases were of this type, and it is possible that some were missed.

Secondly, there was the case in which the more prolonged diarrhœa resembled mild bacillary dysentery.

Gunner C. passed three liquid stools, associated with abdominal pain and tenesmus, during one afternoon. He continued passing three or four fluid stools per day for the next three days, after which the stools became formed. He had no vomiting, and at no time was his temperature elevated.

Thirdly, there was a group of five cases which we regard as comparable to a well-recognized picture of Salmonella infection, chiefly because the symptoms seemed to be largely upper abdominal in type and the fever was more prolonged.

Driver R. gave a history of nausea, abdominal pain and the passage of five liquid stools in the previous twelve hours. His temperature was 101° F. and the pulse rate 120 per minute. Next day he passed two fluid stools with pronounced tenesmus, he vomited on four occasions, his temperature was 102° F. and he looked ill. On the third day he remained febrile and passed one fluid stool. Thereafter his fever abated, the stools became formed and the sigmoidoscopic appearances were normal. On the second day of his illness the leucocytes numbered 8,700 per cubic millimetre, the differential count revealing no abnormality, and an attempt at culture of organisms from the blood was unsuccessful.

Treatment.

Early in the epidemic patients received a routine course of sulphaguanidine, and subsequently all those whose stools contained a cellular exudate were given this drug. We know of no specific chemotherapy for Salmonella infections, and both the sulphaguanidine given in this epidemic and sulphadiazine, which we have tried in other Salmonella infections, had no obvious effect in shortening the illness or shortening the carrier period. No saline purgatives were given.

All patients were investigated for absence of Salmonella organisms from the stools before they were discharged from hospital. Unfortunately we had to be satisfied with

TABLE I.

Salient Clinical Features in Twenty-six Bacteriologically Proven Cases.

Bowel Actions in First Twenty-four Hours.			Duration of Diarrhœa.				Vomiting.		Pyrexia.			Duration of Pyrexia.			
Greatest Number.	Least Number.	Average Number.	One Day.	Two Days.	Three Days.	Over Three Days.	Present.	Absent.	Observed.	Not Observed.	Average Temperature.	One Day.	Two Days.	Three Days.	Over Three Days.
16	2	6·6	7	11	4	4	8	18[1]	17	9	100° F. to 101° F.	11	4	2	Nil

[1] Five of these patients complained of nausea.

one "negative" stool in many instances. When the patients were on our own hospital staff, however, two successive "negative" stools had to be passed before they were allowed to resume duty. In the 26 cases in which the organism was isolated initially, the stools were "clear" within seven to ten days in 14 cases, within twelve to sixteen days in seven cases, within eighteen to twenty-two days in three cases, and after twenty-two days in two cases; in the last two cases the stools were "clear" in forty-five and fifty-eight days respectively.

Bacteriological Investigation.

Of the 26 Salmonella strains isolated, eight were detected by direct plating of the stool on desoxycholate citrate agar. The remaining 18 strains were detected only after tetrathionate broth enrichment. Proteus strains gave surprisingly little trouble, and it was not infrequent to find an almost pure culture of the Salmonella after the plating out of material from the enrichment broth.

The morphological, biochemical and antigenic characters of the organism have been studied in some detail by Miss Nancy Atkinson, of the Institute of Medical and Veterinary Science, Adelaide. Her preliminary report on the 26 cultures is attached as an appendix to this article.

All 26 strains appeared identical by the ordinary laboratory methods which we were able to apply. The organism was a Gram-negative, motile, rod-shaped bacillus growing well on ordinary media. Glucose and mannitol were fermented, with acid and gas production, abundant hydrogen sulphide was formed and indole was not produced. There was no action on lactose or saccharose after three weeks' incubation, nor did fermentation occur in the few strains incubated for longer periods (up to fifty days). With Commonwealth Serum Laboratories polyvalent Salmonella serum, each culture yielded positive slide agglutination, but all failed to react by the slide method with other common Salmonella sera, such as *Salmonella typhi*, *Salmonella paratyphi* A and B, *Salmonella typhi-murium* and *Salmonella enteritidis* (Gaertner).

One strain was used for pathogenicity tests in laboratory animals. Doses of 50,000,000 bacilli injected intraperitoneally produced fatal peritonitis and septicæmia in mice within thirty-six hours. The organism was readily recovered from heart blood at post-mortem examination. After receiving doses of 9,000,000 bacilli, mice survived for seven to twenty-one days, and died with septic lesions, especially in the kidneys. From some of the latter the organism was recovered in pure culture. Doses of the order of 100 bacilli given intraperitoneally to mice had no apparent effect. Guinea-pigs behavied in the same general manner, but were more resistant than mice. A Salmonella (related to *Salmonella reading*) was given in equivalent doses to mice and guinea-pigs, and results were comparable in every way to those obtained with the test organism. Equivalent doses of a fæcal strain of *Bacterium coli* were well tolerated in mice.

Members of the Salmonella group vary greatly in their pathogenicity to laboratory animals, but this feature has not been extensively studied. *Salmonella typhi* is lethal to mice only in doses greater than 50,000,000, whereas *Salmonella typhi-murium* is lethal in doses as low as 100 bacilli.

Unfortunately some strains became partially rough. This was probably due to prolonged incubation in lactose and sucrose, preceded and followed by subcultures on many different media. It seems possible, therefore, that biochemical variants may have arisen.

Serological Investigation.

An O suspension, prepared by Bien's method, was made up from the first organism isolated, and specimens of serum from all 26 patients, obtained between the eleventh and fourteenth days from the onset of the illness, were set up against them. Nineteen specimens showed agglutinins in titres of 1 in 10 to 1 in 320, while approximately half had titres of 1 in 40 or over. Six of the higher titre specimens were tested with a *Salmonella typhi* O suspension and showed no agglutination. Thus the agglutinins were not the result of "T.A.B." inoculations. To exclude the possibility that a significant number of normal people might have these agglutinins, a series of 100 control specimens of serum was tested with our original O suspension. The control series comprised serum received by the laboratory for routine Kline and Weil-Felix tests. Six control specimens gave some agglutination, but none of higher titre than 1 in 20. This suggests that a titre of 1 in 40 or over is probably significant of present or past infection, and is in keeping with the titres commonly found in Salmonella infections of this type. Two specimens which gave a titre of 1 in 160 were excluded from the control series, because both men gave a history of diarrhœa at the time of the epidemic. Subsequent investigation showed that one of them was still excreting the test organism in his stools.

A second sample of serum was obtained from 22 of the 26 patients seven to ten days after the first sample had been collected. With few exceptions the titres were now falling, and in none was the titre rising.

The results of these three series of O agglutination tests are set out in Table II. As a final check, six specimens of serum were tested with O suspensions prepared from eight other organisms isolated during the epidemic. The titres corresponded well to those obtained with the original O suspension.

TABLE II.

"O" Agglutinin Titres in Patients' Serum Compared with Control Series.

Test Group.	Number of Specimens Tested.	Serum Titres.[1]							
		Nil	1/10	1/20	1/40	1/80	1/160	1/320	Over 1/320
Patients' serum, 11 to 14 days from onset	26	7	3..	4	5	1	5	1	Nil
Patients' serum, 21 to 24 days from onset	22	11	1	3	6	1	Nil	Nil	Nil
Control serum	100	94	5	1	Nil	Nil	Nil	Nil	Nil

[1] The figures represent the number in each group.

Epidemiology.

In the early stages of the epidemic, cases were limited to hospital staff and patients. A clinical and bacteriological investigation of cooks and mess orderlies was therefore carried out, but results were completely negative. Later it became evident that at least three soldiers contracted infection outside the hospital area. Thus we were not dealing with a completely localized epidemic. Further inquiries elicited the information that there was a peak in the general incidence of diarrhœa amongst troops in the area at this time, and personal information from regimental medical officers revealed small epidemics in units from which our "outside" cases came. It is possible, therefore, that quite a widespread epidemic actually occurred. A small number of mice were trapped in certain areas of the hospital grounds, and bacteriological investigation of their bowel contents gave negative results. Unfortunately the number examined was too small for this investigation to be really significant.

The known incidence of disease in our hospital area was remarkably low, only 2% of the hospital population being affected. Since some cases were so mild and were detected only by direct questioning, it is likely that many more soldiers suffered mild attacks and did not consider them worth reporting to a medical officer. The true incidence in the hospital area was almost certainly much higher than was apparent. The frequency of the undetected mild case is probably a big factor in spreading infection and prolonging an epidemic, because the patient with a known infection is admitted to hospital and segregated until his stools are free from the organism. Three further cases of this type of Salmonella infection occurred about three weeks after the end of the epidemic; they may have been contracted from an undetected convalescent carrier.

It is questionable whether the observation has any value, but it was noted that approximately half the patients first noticed symptoms in the early hours of the morning—from 2 a.m. to 5 a.m. This would suggest, perhaps, infection at the evening meal and an incubation period of about twelve hours.

Discussion.

In an army camp the presumptive diagnosis in acute diarrhœa is bacillary dysentery, and experiences with an Australian general hospital in New Guinea have borne out the value of this dictum. In the area of the hospital under discussion there is a very low incidence of diarrhœa, and bacteriological proof of its ætiology is frequently not obtained.

We have investigated, under conditions apparently ideal for the isolation of dysenteric organisms, several small epidemics of diarrhœa in the area, and results, as in sporadic cases of diarrhœa, have been singularly disappointing. Sporadic cases of Salmonella gastro-enteritis have been encountered occasionally. On one occasion *Salmonella typhi-murium* was isolated from the stools of two soldiers who shared a tent in their unit and were admitted to hospital with a typical picture of Salmonella infection. On other occasions a Salmonella (related to *Salmonella reading*) has been isolated from stools in nondescript cases of diarrhœa, clinically indistinguishable from the average case with which we were dealing. It seems possible that in this area Salmonella organisms cause a higher incidence of diarrhœa, and dysenteric organisms a lower incidence, than is usual in army camps. We believe that Salmonella infections will be missed unless appropriate enrichment methods are used in the laboratory, and perhaps unless several stools from each patient are examined.

In the epidemic at present under discussion we believe the evidence is good, although necessarily circumstantial, for regarding the organism isolated as being the actual cause of the disease. The most important points appear to be as follows.

1. The organism was isolated from a high proportion of patients—from 26 of the 29 patients bacteriologically investigated. In some cases, early in the illness, we made a daily cultural examination of the stools, and the organisms were constantly present in moderate numbers.

2. The organism disappeared from stools *pari passu* with the recovery of the patients, as is seen from Table II.

3. Over the four months subsequent to the epidemic, stools from 241 patients with diarrhœa have been culturally examined, enrichment methods being used, and this organism was isolated on only four more occasions. There was reason to believe that three of these cases were connected with the epidemic, as they occurred shortly afterwards. In this four-month period three other Salmonellas were encountered—*Salmonella typhi-murium* and a Salmonella related to *Salmonella reading* twice.

4. The serum of a significant number of patients contained O agglutinins to the organism. These appear to be specific antibodies developed as a result of infection by this organism. The fact that this strain has an unusual antigenic structure is further evidence for regarding these antibodies as specific.

5. Finally, the clinical features of the various cases accord reasonably well with a recognized picture of Salmonella infection.

Summary.

1. An epidemic of gastro-enteritis in the area of an Australian general hospital is described in brief.

2. The infections were mild in nature, and the mortality was nil.

3. A new strain of Salmonella organisms was isolated from 26 of 29 patients bacteriologically investigated. The name *Salmonella atherton* is suggested.

4. Evidence in favour of this organism's being the cause of the epidemic is briefly presented.

5. The value of an enrichment broth for the detection of Salmonella organisms in stools is emphasized.

Acknowledgements.

We are indebted to Lieutenant-Colonel R. R. Andrew for his stimulating interest and advice, to Miss Nancy Atkinson for her work in elucidating the antigenic structure of the organism isolated, and to Major-General S. Roy Burston, Director-General of Medical Service, Australian Military Forces, for permission to publish this article.

Appendix.

(N.A.)

This preliminary report contains a brief account of some of the cultural and serological reactions of a group of 26 cultures obtained from fæces of patients in a single outbreak of gastro-enteritis. A detailed account of these organisms will subsequently appear elsewhere.

The organisms were all Gram-negative, motile bacilli, growing readily on ordinary media, not forming indole and promptly fermenting glucose and mannitol with the production of acid and gas. The majority of the strains slowly liquefied gelatin, slowly fermented sucrose with slight acid formation, and after serial transfer in lactose medium slowly fermented lactose also. These reactions are not typical of the Salmonella group.

Antigenically the 26 strains appeared to be identical. They all contained the H antigen z_1 found in a few Salmonellas, but their O antigens were different from any Salmonella O antigens so far described. They also contained the H antigen z_{28}, which occurs with z_4 in one Salmonella (*Salmonella cerro*). This combination of H antigens z_4 and z_{28} also occurs frequently in a group of paracolon organisms described by Peluffo, Edwards and Bruner (1942), and by Edwards, Cherry and Bruner (1943). For lack of better names, I shall refer to this group for the present as the z_4 paracolon organisms and to ours as the "Atherton" strains. Culturally and antigenically (as far as the H antigens were concerned) the Atherton strains closely resembled this group. At present I have only one member of the group with which to compare our strains. It is known as "Arizona", and, according to Edwards *et alii* (1943), has the antigenic formula XXXIII, B, E:$z_4z_{23}z_{28}$. Neither the O nor the H antigens of the Atherton strains were identical with those of Arizona. I shall shortly receive from Dr. Edwards the other main types of the z_4 paracolon organisms, and a complete antigenic comparison between these and the Atherton cultures will establish their relationship.

With the exception of one, the z_4 paracolon organisms of Edwards *et alii* (1943) were obtained from animals, and there was strong epidemiological evidence that they were the ætiological agents of animal disease. One strain was

obtained from man, but its history was unknown. This strain, and one obtained by Seligman and Saphira (personal communication from Dr. Edwards) from a fatal infection in man, are the only two human strains recorded. The role of these organisms in human disease therefore requires elucidation. The present report provides the first solid contribution to the evidence for their pathogenicity for man.

The z_4 paracolon organisms are closely related to the Salmonellas, and could be included in this group according to the definition of the Salmonella Subcommittee of the International Association of Microbiologists (1941), which allowed the introduction of occasional lactose-fermenting and gelatin-liquefying types. The indiscriminate inclusion of paracolon types in the Salmonella group should, however, be discouraged, as atypical coliform organisms of doubtful pathological significance would undoubtedly creep in. When, however, the pathogenicity of a paracolon type is established beyond reasonable doubt, it could, with advantage, be admitted to the ranks of the Salmonellas. The Atherton cultures appear to be human pathogens producing a typical gastro-enteritis. By ordinary procedures they would not be classed as lactose-fermenters, and their fermentation of sucrose is slow and slight. In my opinion they could be placed in the Salmonella group as a new type *Salmonella atherton*, for which the antigenic formula has not yet been fully worked out.

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A CASE OF PNEUMOCOCCAL MENINGITIS ASSOCIATED WITH CHRONIC MASTOID INFECTION AND COMPLICATED BY MALARIA.

By JOHN H. COLEBATCH,
Major, Australian Army Medical Corps.

Clinical Record.

THE patient, a sapper, aged thirty-four years, was admitted to an Australian casualty clearing station in the evening of October 12, 1944. He stated that he had developed a slight "cold" a week previously. He had complained of anorexia, headache and abdominal discomfort on October 11, and of a more severe attack with shivering and a temperature of 101·4° F. on October 12, but he had not vomited. He had had six attacks of malaria, the last of them in June, 1944. He had taken no "Atebrin" since July.

On examination, the patient was emaciated, the temperature was 99·8° F., the spleen was palpable and tender. No malarial parasites were seen in a blood film. Next morning the headache was worse and he refused food. The temperature was 97° F., the pulse rate 68 per minute. By 11 a.m. he was restless and confused and showed signs of meningeal irritation. No other abnormality was found except some faucial injection and wax in the right ear. Cerebro-spinal fluid from the lumbar theca was opalescent and under increased pressure, and examination of the fluid showed that the total protein content was more than 300 milligrammes per millilitre, the leucocytes numbered 4,000 per cubic millimetre, all being neutrophile cells, and both intracellular and extracellular diplococci were present in very large numbers. Culture of the fluid produced in twenty-four hours a profuse growth of pneumococci, as penicillin-sensitive as the average hæmolytic streptococcus. Treatment was begun at noon with penicillin, 15,000 units being given intramuscularly every two hours, and a continuous intravenous drip administration of glucose and saline solution was instituted. At 3 p.m. penicillin diluted with *aqua destillata* was given intrathecally, 10,000 units in four millilitres. The cerebro-spinal fluid collected at this time contained 6,000 leucocytes per cubic millimetre. The patient was extremely irritable, verging on coma; the rectal temperature was subnormal (97° F.) and the pulse rate 64 per minute. A catheter specimen of urine contained much sugar, a trace of acetone, and no albumin, and microscopic examination revealed no abnormality. The leucocytes numbered 30,000 per cubic millimetre, of which 28,500 were neutrophile cells. No malarial parasites were detected in a second blood film. By 10 p.m. his condition was grave. He was in deep coma, with irregular breathing of Cheyne-Stokes type; the rectal temperature was 101·4° F.; the pulse rate had fallen to 56 per minute. A concentrated magnesium sulphate enema was given, and two grammes of soluble sulphapyridine were administered parenterally. Two hours later his condition began to improve slightly and the respiratory rhythm was normal.

At 8 a.m. on October 14 the clinical picture had altered remarkably. He was stuporose, but far less irritable; his temperature, pulse rate and respirations were normal, and he began to take sips of water. He was still incontinent of fæces and had urinary retention. A catheter specimen of urine contained much sugar, but no acetone. Sulphapyridine (two grammes) was given parenterally, and saturated sodium bicarbonate solution (ten millilitres) and 4 molar sodium lactate solution were administered intravenously. The cerebro-spinal fluid contained 12,000 leucocytes per cubic millimetre, but no organisms. Penicillin (10,000 units) diluted with cerebro-spinal fluid was given intrathecally. By the evening he was able to take alkalis and sulphamerazine (four grammes) twice a day by mouth.

On October 15 he commenced to talk, to take adequate fluids by mouth, and to void urine voluntarily. The number of leucocytes in the blood and the cell count in the cerebro-spinal fluid had fallen considerably. Penicillin treatment was continued intrathecally (10,000 units) and intramuscularly (15,000 units every three hours). For twenty-four hours a blotchy erythema appeared on the buttocks, the site of most of the injections.

The patient's condition continued to improve satisfactorily, and he was fully conscious and rational by October 18. Photophobia and restlessness disappeared after several days, and neck stiffness and Kernig's sign after another week. The tendon jerks in the legs were the last to recover. The accompanying chart (Figure I) demonstrates the main points

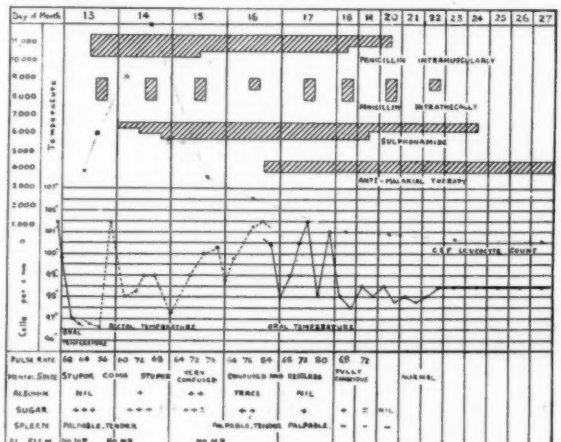

FIGURE I.

in his treatment and progress. The number of leucocytes in the cerebro-spinal fluid fell gradually to 23 per cubic millimetre on October 26. On two occasions the intrathecal dose of penicillin was reduced to 5,000 units, but the concentration was not altered. The penicillin content of the cerebro-spinal fluid was estimated by the agar cup method on October 17 and 18, and on both days it was less than one unit per millilitre. There was an indefinite response to the Queckenstedt test on October 18 and no response at all at the last lumbar puncture on October 22. By December 5 the patient's recovery from the meningeal infection was complete except for some general weakness and emaciation and slight impairment of the ankle jerks. He was steadily improving.

There was a recurrence of pyrexia on October 15 and 16, associated with headache and a tender, palpable spleen. No abnormality was seen in a skiagram of the chest. As the meningitic condition was then subsiding, this pyrexia was

regarded as due to malaria. The characteristic clinical response to "Atebrin" supported this contention.

On October 20 earache and signs of inflammation in the right ear developed, with a sanguino-purulent discharge, all of which subsided in three days. The patient then admitted he had been deaf in that ear for years, and had had treatment for earache in March, 1943, and January, 1944. Skiagrams revealed slight mucosal thickening in the antra, and old chronic right-sided mastoiditis with central destruction of bony tissue. An otologist found evidence of an old radical mastoidectomy on the right side, performed probably in infancy. There was gross reduction of bone conduction and no useful air conduction on that side, and the response to Weber's test was to the left. The patient was evacuated to a base area because of his ear condition.

Comment.

This case is reported because of the following interesting or instructive features.

1. The infection was fulminant, unusually large numbers of organisms and neutrophile leucocytes being found in the cerebro-spinal fluid. This presumably explains why the patient's clinical condition continued to deteriorate until nine hours after the first intrathecal dose of penicillin, twelve hours after the first intramuscular dose. Fortunately the condition responded well without recourse to the intraventricular route of administration. The abnormal Queckenstedt response, presumably indicative of intrathecal adhesions, was not present until the second week and did not preclude a satisfactory clinical recovery.

2. Penicillin was given intrathecally, apparently without ill effect, in a concentration (2,500 units per millilitre) five times greater than that which is now generally recommended. It is possible that the rather persistent pleocytosis in the cerebro-spinal fluid was due to the concentrated penicillin, but no harm seemed to result from this feature.

3. The total amount of penicillin given was large—70,000 units intrathecally and 900,000 units intramuscularly. An equally good result would probably have been obtained with smaller dosage. It was found that the concentration of penicillin in the cerebro-spinal fluid twenty-four hours after an intrathecal dose was no higher after 10,000 units than after 5,000 units. The preparations used were "Dri-set" (John Wyeth and Brother) on October 13 and 14, and "Commonwealth" (Commonwealth Serum Laboratories) on subsequent days. The dosage of sulphonamide (65 grammes in eleven days) was deliberately kept submaximal because the good response to penicillin was manifest before an appreciable amount of sulphonamide had been given.

Acknowledgement.

This report is published with the permission of the Director-General of Medical Services. Appreciation is expressed of the assistance received from Major P. de Burgh and from Captain A. Bryson.

Addendum.

This patient wrote spontaneously on June 3, 1945, to say that he was working on his farm again and that he was back to normal weight. He stated: "I have never felt better in my life."

Reviews.

SOCIAL MEDICINE.

FOR some medical students public health is not a fascinating subject. It lacks the thrill and glamour of some of the other studies. Yet the place of medicine in modern society becomes increasingly important, and in these days, more than ever, it is a good thing for every doctor to know at least the trends of development in public health. A few years ago Professor J. R. Currie, of Glasgow, produced an excellent book on the subject, and now a second edition has appeared.[1] On this occasion Professor Currie has had the assistance of Dr. A. G. Mearns, Senior Lecturer in Public Health in the University of Glasgow. This new edition is even better than the old. It is slightly larger—408 pages instead of 308—and it has 89 figures compared with 34.

The big change in the teaching of public health in the medical course came just before the war. In 1938 the General Medical Council in England put forward a new syllabus for hygiene in the medical curriculum. It was designed to meet the need of general medical practitioners. It brought into prominence the modern developments in preventive medicine.

Public health has gone through an interesting evolution. Until about fifty years ago attention was centred mainly on the environment rather than the man. From the "drains and stinks" era, it became focused on the man himself. The idea of preventive medicine was now firmly implanted and was expanding. Not only were infectious illnesses considered, but illnesses of every sort were viewed from the standpoint of prevention. Students in the wards were being taught to ask themselves the question in every case of illness: how might this trouble have been prevented? Now in recent days has come the still wider development of social medicine, dealing with the many aspects of community life in which medical knowledge can bring enlightenment and benefit. What nobler aim can medicine have?

The wider development of public health received an impetus in England following the introduction of national health insurance over thirty years ago. The Ministry of Health and the local health authorities have year by year played an increasingly important part in the Britisher's health. Moreover, the medical profession has been steadily brought into closer relation with the State, and the medical course reflects the trend. The present course of study laid down for students by the General Medical Council includes, in addition to the old-time public health features, consideration of the powers and duties of health authorities, the various aspects of national nutrition, industrial hygiene and infant and maternal welfare.

The book of Currie and Mearns is delightfully written. Its clarity and directness are appealing. It is well integrated, as a good book should be. It is smooth and easy in the reading. It traces in a brief way the history of social medicine. It urges students to make a "social diagnosis" in every case, bringing for consideration at the bedside all the features of heredity and environment which may bear on the illness. The chapters on industrial hygiene and toxicology, vital statistics, food and nutrition, and international health relations are exceptionally well done. There are interesting summaries and commentaries on such recent developments as the White Paper of February, 1944, on national health services; the White Paper of September, 1944, on national insurance; and the Goodenough report on the organization of medical teaching.

Public health varies in its range and activity. In some countries the developments have outrun those in other places. The methods also vary. For the Australian student, modifications must be borne in mind in regard to the legal aspects and with the administrative features. Any book on public health, as pursued in other countries, has its limitations when detailed local rules are sought. However, as a general guide on social medicine and as a record of the present organization and methods now in vogue in the United Kingdom, the book of Currie and Mearns excels anything now available.

[1] "Manual of Public Health Hygiene", by J. R. Currie, M.A. (Oxon.), M.D., LL.D. (Glasgow), D.P.H. (Birmingham), F.R.C.P. (Edinburgh), and A. G. Mearns, B.Sc., M.D., B.Sc. (Public Health), D.P.H. (Glasgow), F.R.S. (Edinburgh); Second Edition; 1945. Edinburgh: E. and S. Livingstone Limited. 8¾" × 5½", pp. 448, with 89 illustrations. Price: 21s. net.

THE SURGERY OF ABDOMINAL TRAUMA.

AN excellent little book sets out in very compact style the experiences and impressions of Major Geoffrey E. Parker, R.A.M.C., during his duties with the army in North Africa, Sicily and Italy.[1] The notes were written as the casualties were received and so are of great value as immediate records. There is hardly an abdominal wound for which treatment is not described briefly and clearly. The chapter on diagnosis deals with types of wounds and missiles and the value of certain procedures; the author condemns attempts to find shifting dulness. Auscultation of the abdomen is invariably carried out, and the value of proctoscopy and catheterization is stressed. The time to operate is often a more difficult decision to make than the need for surgical interference. A chapter is devoted to pre-operative treatment and resuscitation as well as to the principles of post-operative care. This is a very valuable book for the emergency surgeon.

[1] "The Surgery of Abdominal Trauma", by Geoffrey E. Parker, M.B., B.Ch., F.R.C.S. (England), with a foreword by Colonel J. M. Weddell, C.B.E., F.R.C.S.; 1944. London: J. and A. Churchill Limited. 8" × 5¼", pp. 128, with ten illustrations. Price: 10s. 6d.

The Medical Journal of Australia

SATURDAY, DECEMBER 1, 1945.

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SPECIAL ANNOUNCEMENT.

The last issue of this journal was published on September 15, 1945. The hiatus since that date has been due to a dispute in the printing industry in New South Wales which led to the suspension of all work. The issue of September 15 was Number 11 in Volume II, 1945. The present issue is therefore designated Numbers 12-22 inclusive.

THE HEALTH OF ADOLESCENTS.

To say that the present is the day of the young man is to proclaim the obvious. It is true that the ship of State has had at its helm statesmen who could with no stretch of the imagination be called young. But these same statesmen were able to achieve their success because of qualities which most young men can acquire, at least in some measure, if they have the foreknowledge and the wisdom to guide them in the acquisition. One has only to think of the feats of the Royal Air Force in the Battle for Britain to realize the tremendous debt that the people of Britain and of Greater Britain will owe for all time to the fire and intrepid valour of youth. In the face of the example of these men and of so many others like them, it is not surprising that attention has in many places been directed to the health and general efficiency of the adolescent members of the community. As a matter of fact there can be very few who will deny that the hope of the world rests on the physical, intellectual and moral worth of its young men and young women.

Looking back a decade or two in the history of Australia, we recall the system of universal training with its regular military drill and physical betterment. Some members of the community thought this training was good for the youth of that day both from the physical and also from the community point of view, to say nothing of the knowledge of military matters acquired by the boys and its value in the event of a national emergency. Others did not agree with the element of compulsion that encompassed the whole arrangement. Eventually, as is well known, the government abolished the system. With this in mind it is of interest to read the reasons stated by Brigadier Frank Howitt why the achievement and maintenance of health in adolescents made no popular appeal before the war. He was speaking at a discussion on "the development of health in adolescents" at a meeting of the Section of Medicine of the Royal Society of Medicine.[1] The first reason that he stated was the political factor—anyone outside the fighting services who came forward with a demand for national physical fitness was likely to lay himself open to a charge of militarism. The adolescent, Howitt states rather aptly, had no political foster parent—he lived in the no man's land between the realm of the Board of Education and that of the Ministry of Labour. The Ministry of Health apparently was a long way off. Howitt's second reason is the economic factor—"the increasingly long hours of work demanded by the progressive but predatory incursions of education and of industry into the life of adolescents left them neither the time nor the inclination for self-improvement". Thirdly, "there was no motive through which to make an appeal to a freedom-loving country such as ours". Howitt's fourth reason not only states why something did not happen, but points in the right direction for the future—"the development of health in youth is a social issue in which there must be co-ordination between medical science in all its aspects, and all non-medical services interested in physical education, using the term in its widest sense, and not in the restricted form by which it is commonly understood, particularly by the medical profession". The only alternative to compulsion in the matter of physical fitness is persuasion, and the Commonwealth Government, recognizing this, started its National Fitness Campaign in 1938. This campaign followed the adoption of a resolution by the National Health and Medical Research Council in favour of the establishment of an organization "which shall have as its main objective a standard of physical fitness such as this country, with its racial heritages, natural environment and economic opportunities should show". The Council recommended that the Commonwealth Minister for Health should form immediately a National Council for Physical Fitness under the Minister for Health, to invite the State Governments through their ministers for health to form State Councils and to extend the invitation to include local government authorities. Subsequently several conferences were held at Canberra, Melbourne and Sydney. The pursuit of physical fitness in Australia was before long established on a sound basis. In July, 1941, the *National Fitness Act* made the Commonwealth Council for Physical Fitness a statutory body for the purpose of advising the Minister of Health with respect to the promotion of national fitness, and in particular in relation to:

(*a*) the measures to be adopted to develop appreciation of the need for physical fitness;
(*b*) the provision of facilities for instruction in the principles of physical education;
(*c*) the organization of all movements, and the provision of facilities for attaining or maintaining physical fitness; and
(*d*) the training of teachers of classes, and of leaders of movements or groups, formed for the purpose of promoting physical fitness.

The act also provided for a trust account to the known as the national fitness fund. Physical setting up of the individual was not the only objective of the Commonwealth Council for Physical Fitness. It visualized also the establishment of community centres which were to have the following functions: (*a*) the gathering together in a

[1] *Proceedings of the Royal Society of Medicine*, April, 1945.

common meeting place of the members of a community for their common welfare; (*b*) the establishment of a centre for the dissemination of advice and knowledge in all matters of interest to the community; (*c*) a social and recreational centre for all members of the community. The Physical Fitness Campaign is intended to cover members of the community of all ages, and not only adolescents. However, the adolescents are not left, as in England, in a "no man's land"; the government departments of education and of labour are not their nearest approach to a foster parent—they are entrusted to the Ministry of Health, where they rightly belong. At this stage it is important to note that at its seventh session in September, 1943, the Commonwealth Council for Physical Fitness declared that though the events of the previous two years had necessitated the restriction of its activities to the limited field of physical education, it was desirable to revert to the original concept of national fitness on a wider basis "with the object of encouragement, through recreational and other community activities, of a strong spirit of community service".

It is interesting to note that, according to Brigadier Howitt, the British Ministry of Health, in conjunction with the Ministries of Education and Labour, is considering the initiation of a civilian centre for substandard youths. This must not be confused with the type of community centre discussed in connexion with the Australian body. Howitt points out that since compulsion cannot be used, the success of persuasion is dependent upon motive. The motive suggested by him is based on realization that we live in a competitive world, and that, democracy or no democracy, everyone must fend for himself. "The appeal", he states, "must therefore be pragmatic and practical, even selfish, and based on the lure of heightened individual efficiency, with resultant self-advancement and an increased earning capacity and prestige." This is an appalling and most regrettable statement; it represents a point of view which, universally adopted, would bring nothing but disaster on the community, on the nation and on civilization itself. Man is selfish enough by nature and needs more than anything else a motive that will turn his thoughts and desires away from his own appreciation and aggrandisement. The idea of community betterment that is incorporated in the Commonwealth National Fitness movement is much more commendable than Howitt's unworthy suggestion, for it does imply an element of service by the individual. What must be recognized is that the health of an adolescent, or of anyone else for that matter, is not attained if he develops a sound and healthy body, capable of endurance and of withstanding adverse conditions, and the activities of that body are not under the control of his mind and will. The development of intellectual and moral qualities is just as necessary to the fitness of a man as is the cultivation of the body, strong and beautiful. And, of course, moral and intellectual qualities can be cultivated just as surely as healthy organs and big muscles, particularly if a start is made in the very early years. Here we may digress and remind ourselves that, as emphasized by Alexis Carrel, the same factors that determine the shaping of the young human being are able to prevent the deformation of the old. A wise use of psychological influences retards the decay of many men. This without doubt has something to do with the helmsmanship of the statesmen, not so young in years, that has already been mentioned.

On the purely physical side several present-day tendencies must be mentioned only to be condemned. First and foremost is the second-hand enjoyment of sport and of outdoor excise by the enormous crowds that gather to watch sporting fixtures. The people who benefit most are the players; the onlookers are satisfied with the emotional thrill of seeing a game well played or the excitement of barracking for a side of which they are partisans. It must be admitted that on the whole Australians do not fare badly in the matter of out-of-door exercise, but it would be of great advantage if more playing fields were provided in certain congested areas, even by the resumption of properties for the purpose. Physical education of adolescents must not be allowed to start only when the stage of adolescence has been reached. It should start with the play of the school and the pre-school child. In this regard it would be advantageous to adolescents of the future if Saturday afternoon picture shows for small children were prohibited by law. The fact that cinema houses are crowded by children every Saturday afternoon is a standing disgrace to a people so privileged and gifted with sunshine as the people of this land are. Again the adulation given to popular sporting heroes is thoroughly bad—it is bad for the heroes and bad for the adulators. Sir Adolphe Abrahams made a good point in the Royal Society of Medicine discussion. He was dealing with the current fallacy that excessive sport might damage healthy cardiac muscle—the great athlete acquired a wrong sense of values from the adulation that was accorded him, and Abrahams was disposed to submit that the chief danger to the adolescent was not an enlarged heart, but an enlarged head. Other aspects of this subject might be mentioned, but it will suffice to conclude by quoting G. E. Friend, who said at the London discussion that the development of health in adolescence depended on: (*a*) the provision of an adequate and well-balanced diet; (*b*) the provision of adequate sleep and rest; (*c*) a properly balanced physical development, which in turn depended on a correct ratio between games, physical training and education in personal and environmental hygiene; (*d*) avoidance of over-fatigue, mental as well as physical. Such statements are useful as a general guide. Individual adolescents of both sexes will often need individual treatment. There may on the one hand be cases in which the training of the body should be curtailed or in which there should be no insistence on the participation in organized sport. On the other hand it is a good general rule that growing children should not be pampered. The decision should not rest finally with the child, and the person who has to make it must deal with every aspect of the child's make-up.

Current Comment.

SPONTANEOUS PNEUMOTHORAX AS A COMPLICATION OF PNEUMONIA.

PNEUMOTHORAX may be caused by erosion or spontaneous rupture of lung tissue. It may occur during excessive exertion, as, for example, during a fit of coughing, especially if the lung is emphysematous, or during a paroxysm of coughing in pertussis. It may follow the softening and breaking down of a septic infarct or it may be produced in a minute bronchiectatic cavity or in con-

nexion with a tuberculous lesion. An unusual form of pneumothorax is that caused by the gas formed by micro-organisms during life—the type of pneumothorax resulting from gunshot and other wounds of the thorax. A type of pneumothorax, not often recognized, occurs in the course of pneumonia. This interesting phenomenon has been the subject of comment by E. R. Movitt,[1] who reports two cases and comments on the rarity of the condition, since only twenty cases have been recorded in the literature. Two points emerge from his report—the first that the occurrence of this lesion may lead to the unwarranted suspicion of tuberculosis, the second, that the lesion is not recognized as often as it should be because the pneumonic symptoms mask it and X-ray examination which would reveal it is not undertaken. The first of Movitt's two cases was that of a man, aged forty-three years, who suffered from pneumonia that responded to sulphonamide therapy. An apical pneumothorax was discovered on routine X-ray examination. In spite of examination by every method, including guinea-pig inoculation, no evidence of tuberculous infection was discovered. In Movitt's second case, the patient, a male, was forty-nine years of age. The pneumonic process was severe and affected the right lung. Temporary improvement took place and subsequently physical examination revealed signs suggestive of a hydro-pneumothorax or pyopneumothorax; the suspicion was confirmed on X-ray examination. Repeated thoracocentesis yielded clear fluid in which the presence of organisms could not be discovered, by examination of a smear preparation, by attempts to produce a culture or by guinea-pig inoculation. No organisms were recovered in culture from the blood. The patient's condition rapidly failed and he died twenty-two days after admission to hospital. At autopsy the lung was found to be "partially collapsed and adherent along its posterior surface to the chest wall by moderately dense adhesions". The dull and granular surfaces of the lung were covered with fibrin and presented the usual appearance of bronchopneumonia. The bronchial mucosa was congested and the bronchi were partially filled with purulent exudate. The pulmonary vessels were patent. There is no information to show where or how the pneumothorax was produced. Signs of acute bacterial endocarditis were found. It is remarkable that no organisms were found in the pleural effusion in spite of the most careful search.

From a review of the scanty literature and from his two cases Movitt thinks that spontaneous pneumothorax in association with pneumonia may be classified into two varieties—the first, with or without serous effusion and presumably sterile, the second associated with empyema. The first variety is a comparatively innocuous complication. Movitt discusses the work of Pierce and Dirkse on pulmonary pneumatocele and thinks that this condition may be an antecedent to the pneumothorax, which is of the closed variety. The second variety of pneumothorax is open and may quickly evolve into pyopneumothorax. The pathogenesis is, he states, easily understandable, as the situation resembles that of a pulmonary abscess or tuberculosis. Both varieties may be masked by the grave symptoms of the underlying pneumonic process.

[1] *The American Journal of the Medical Sciences*, May, 1945.

OXYGEN TOXICITY.

In 1878 Paul Bert announced that hyperoxygenated air at increased pressures produced convulsive seizures in dogs during or following decompression, and this reaction he attributed to the formation of some toxic chemical substance arising from the oxygen excess. Leonard Hill subsequently pointed out that in Bert's experiments the decompression was rapid and that though the danger with oxygen alone is far less than when nitrogen is present, effervescence could easily occur. Further researches, however, showed that, with decompression so slow as to exclude all possibility of gas embolism, toxic conditions still arose. John W. Bean and Ernest Siegfried, of the Department of Physiology in Ann Arbor, have recently subjected young unanæsthetized rats to oxygen at "65 pounds pressure" (*sic*) in short single or successive exposures.[1] The reactions observed during the decompressional and post-decompressional periods could be classified as acute and chronic. The acute showed various manifestations of central nervous system involvement such as stupor, coma and motor dysfunction, both tonic and clonic. The chronic type was characterized by spastic paralysis limited to the forelegs in less pronounced cases but affecting the whole musculature in the severe cases. These chronic effects, though capable of some amelioration, were predominantly permanent. The authors attribute these toxic conditions to accumulation of endogenous carbon dioxide in the tissues, increased tissue acidity, some interference with tissue enzymes and also definite injury to nerve cells, particularly those in the upper motor neurones. In these investigations and in many others on the same theme there does not appear to be any proper realization on the part of the experimenters that cellular activity is primarily anoxybiotic. Oxidation comes later to reenergize the mechanism or to act in a depolarizing capacity. Just where the frontier of oxygen lies in the living cell we do not know, but it cannot be far away from the cell wall. When oxygen under higher tensions than normal is supplied to the living cell, it is driven into regions where it is not wanted and where it can do harm. As an instance of what Henderson called "the fitness of the environment", we find that our bodies are attuned to breathing air in which the oxygen pressure never exceeds 159 millimetres of mercury; for higher pressures there is no adjustment, as such pressures were never experienced in the course of evolution.

A research of very different character and with immediate clinical application is that of Julius H. Comroe, Robert D. Dripps, Paul R. Dumpke and Margo Deming on the effect of inhalations of high concentrations of oxygen at normal air pressure.[2] Ninety healthy young men between the ages of nineteen and thirty-one years were subjected to atmospheres enriched with oxygen and were kept under continuous observation for twenty-four hours. Control experiments with the same apparatus, but with the subjects breathing ordinary air, were also carried out. The authors correctly emphasize the fact that with tent or catheter the oxygen reaching the lungs is never above 50%, and this they declare to be harmless. Higher concentrations produced a number of signs and symptoms among which substernal distress was almost invariably present. There were also noted coryza, sore throat and a fall of vital capacity. Breathing 100% oxygen had no ill effects at high altitudes or if the period at sea level was very short. These authors wisely state: "We feel that the clinician must bear in mind that oxygen is a drug and must be used in accordance with well recognised pharmacologic principles; i.e., since it has certain toxic effects and is not completely harmless (as widely believed in clinical circles) it should be given only in the lowest dosage or concentration required by the particular patient." They make the pertinent observation that whilst the physician of today insists on the carrying out of quantitative analyses of many blood constituents, he practically never bothers to learn what the oxygen saturation of the arterial blood really is, and it is this criterion which should determine his decision to administer oxygen, at what concentrations and for how long. They give three chief groups of indications for oxygen therapy: (i) to combat arterial anoxæmia; (ii) to hyperoxygenate the blood in conditions not associated with anoxæmia such as coronary occlusion, fever, polycythæmia and so on; and (iii) to eliminate nitrogen from the body in order to prevent decompression sickness or to relieve intestinal distension. In dealing with anoxæmia they make the very common omission of all reference to the removal of carbon dioxide, which is just as important as the supplying of oxygen. The whole paper is a valuable one and should be read in its entirety.

[1] *American Journal of Physiology*, May, 1945.
[2] *The Journal of the American Medical Association*, July 7, 1945.

Abstracts from Medical Literature.

GYNÆCOLOGY.

Intraepidermal Cancer of the Vulva.

T. N. A. JEFFCOATE, T. B. DAVIE AND C. V. HARRISON (*The Journal of Obstetrics and Gynæcology of the British Empire*, October, 1944), working in the Department of Obstetrics and Gynæcology and Pathology of the University of Liverpool, have described a rare condition of intraepidermal cancer of the vulva. It is known by two names—Bowen's disease and Paget's disease; but the authors prefer to use the title of Bowen's disease. Clinically the condition shows as a dull, raw, red area with a clearly defined serpiginous outline. The area is on the same level as the surrounding skin, or at the most is only slightly raised, but there is a sharp line of demarcation between healthy and diseased tissues. The surface is moist, but may become encrusted in part, the appearance being very similar to that of Paget's disease of the nipple. It does not show any of the usual characteristics of malignant disease. There is no friability or fixation, and metastases in lymphatic glands or elsewhere are rare. It may be confused with eczema or with tuberculosis or other ulcers. It is sometimes mistaken for kraurosis or leucoplakia, which may occur in association with Bowen's disease. The disease runs a slow course, often lasting many years. It may at any time change into epithelioma. The symptoms produced are pain and pruritus, the area often being extremely tender. In long-standing cases a history of remissions and exacerbations is the rule. The authors report two cases in detail. The most effective treatment is excision of the part. The article contains a full description of the pathological changes, together with a series of histological studies of the condition.

Anaphylaxis of Menstrual Origin.

B. J. BASILIOU (*Revue médicale française du Moyen-Orient*, October, 1944) reports two cases of severe anaphylaxis of menstrual origin. Both patients had suffered from tuberculosis; the first appeared clinically and radiologically cured, and the second had been left with a tracheotomy opening, but otherwise her tuberculous condition appeared to have been controlled and she had gained considerably in weight. In both cases the symptoms of anaphylaxis were indisputably connected with the menstrual periods. In the first case they began with general malaise, a few hours before the onset of menstruation, and were followed by injection of the bulbar conjunctiva, sensations in the skin that seemed to come from internal burning, "ferocious" pruritus, and the appearance of eruptions of irregular outline and varying shapes, at first papular, red or white, like a nettle rash and with migratory tendencies, and later fixed, single or confluent, and in certain sites reaching the size of the palm of the hand. These eruptions quickly filled with serous or sero-sanguineous fluid. The picture resembled that of urticaria, and the eruptions varied from the vesicular or bullous type to the hæmorrhagic and gangrenous type. At times the condition gave rise to anxiety, suggesting pemphigus or Duhring's dermatitis, and about the second or third day of the menstrual period some of these lesions ruptured, revealing advanced destruction of the skin and with the production of intolerable pain. In the second case somewhat similar signs and symptoms were accompanied by threatened asphyxia from endolaryngeal and tracheal œdema, resistant to all treatment. Careful investigation failed to reveal any parasitic or other cause for the condition, and in the first case ten different forms of treatment produced no benefit. At last desensitization by the patient's menstrual blood was undertaken and effected a complete cure. In the second case this treatment was refused on the grounds of repugnance and religious objections, and the patient died from asphyxia during an attack which occurred on the first day of her last menstrual period. The author describes in detail the preparation of the serum and the method of desensitization.

Trichomoniasis.

ROBERT B. GREENBLATT (*The Western Journal of Surgery, Obstetrics and Gynecology*, May, 1945) has reviewed the literature and concludes that there is still much confusion as to the incidence, pathogenicity, therapy and status of *Trichomonas vaginalis*. Many women harbour the organism without complaining of pruritus or an unusual vaginal discharge. When the condition is acute the discharge is copious, thin and yellow, and contains bubbles, and the mucosa is extremely reddened and "strawberry-like"; but in chronic cases the mucosa may appear normal. The incidence varies greatly. In some venereal clinics it rises to 24%. In a group with proved gonorrhœa, trichomonas was found in 62·5%. Changes in the vaginal fluid enhance the growth of the organism. It will not endure in the secretions of the vagina with normal epithelial maturation and acid pH; but at the same time it is often found both in the acid vagina of pregnancy with heavily glycogen-laden cells and also in the alkaline vaginal secretions of the menopausal woman with immature and epithelial cells with a poor glycogen content. The results of therapy vary very much. There are many excellent preparations capable of ridding the vagina of the organism in three to fourteen days, but the recurrence rate is often distressingly high. In the chronic carrier treatment is much less efficacious. Sulphanilamide and penicillin will clear up concurrent gonorrhœa, but not the trichomonas infection. The author has tried silver picrate insufflations and suppositories, capsules of ozonide of olive oil, sulphathiazole β-lactose tablets and insufflations and "Stovarsol"-medicated tampons, all with satisfactory results. Failure in therapy he attributes to the following factors. (i) The medication did not reach the infected urethra, which proved a source of the persistent reinfection. (ii) The cervix is possibly a latent focus, for with the onset of menstruation a few organisms are set free and rapidly multiply and reinfect the vagina. (iii) Reinfection occurs on resumption of marital relations. Trichomonads may be demonstrated, though very few in numbers, in the urethral and prostatic secretions of 5% to 10% of men. The author recommends treatment of the infected cervix by mild cauterization, urethral irrigation with mild non-irritating antiseptics such as acriflavine (1 in 1,000) or "Zephiran" (1 in 2,000), and acid douches for the vagina—two or three tablespoons of vinegar to a quart of warm water twice a day for three months and two or three times a day during menstruation. During menstruation medicated tampons should be inserted into the vagina. The author recommends medicated tampons dusted with 1·5 grains of "Stovarsol" and having attached to the tip a tablet made of 0·5 grain of "Stovarsol" and 2·0 grains each of dextrose, lactose and boric acid. These medicated tampons have proved most satisfactory and convenient in the acute stage, and also as a prophylactic measure in the chronic recurrent case. Whether the trichomonad is a pathogen or a saprophytic organism requires further consideration. The evidence available favours the latter view. Pruritus seems more common in some women than in others. A carrier stage may occur after repeated attacks and in infections such as those of prostitutes, when the trichomonas proves of little or no inconvenience to the patient.

The Prevention of Cancer of the Cervix.

B. Z. CASHMAN (*American Journal of Obstetrics and Gynecology*, February, 1945) discusses the results obtained in 10,000 cases in which deep cauterization of the cervix was carried out and which were reported in 1941. In the follow-up 4,487 patients have replied to a *questionnaire*. The author has formed the opinion that chronic cervicitis is a contributing factor in the causation of carcinoma of the cervix. Cancer of the cervix is insidious in onset, and because of the late stages in which it is seen today, prevention of cervicitis, prevention of cancer by adequate treatment of existing cervicitis and early diagnosis by periodic examination of women over twenty-five years of age, offer the best solution of the problem. In order to destroy infection in the cervix by cauterization, it is often necessary to cauterize deeply and extensively. Care must be taken post-operatively to prevent stenosis. As far as the author is aware only two cases of cancer of the cervix have occurred in the series of 10,000 patients.

The Spasmolysant Action of Magnesium Ions on the Tetanically Contracted Human Gravid Uterus.

A. R. ABARBANEL (*American Journal of Obstetrics and Gynecology*, April, 1945) has investigated the evolution of a therapeutic agent which would effectively and efficiently exert a spasmolysant effect upon the myometrium. Such spasms occur in so-called spasmodic dysmenorrhœa and in after-pains in many multiparous women, and also in the induction of labour by oxytocics, especially posterior pituitary extracts. After reviewing the pharmacological history of the magnesium ion which many workers have shown will relax spasm in contracted uterine muscle, the author and his co-workers undertook work on patients

in normal labour and estimated the effect on the human gravid uterus of various oxytocic drugs, using external hysterography as a control. They worked out the patterns of human uterine motility produced by various drugs and tested the effect of magnesium ions, giving ten millilitres of a 20% magnesium sulphate solution intravenously. Before the needle was withdrawn from the vein the tetany of the uterus was completely abolished and the uterus continued to contract intermittently and rhythmically. The pain was completely relieved in sixty seconds. A number of graphs of the effects obtained are shown. The author concludes that the magnesium ion exerts an immediate spasmolysant effect on the human gravid uterus, whether caused by pituitrin, pitocin or pitressin, quinine, ergonomine or metherzine, by intravenous administration of two millilitres of a 50% solution of magnesium sulphate or ten millilitres of a 20% solution of magnesium gluconate. It acts probably on the myometrium, slowing the contraction wave. The ion has no demonstrable effect on the pattern of uterine mobilization in the first stage of labour, but it does exert a definite analgesic effect on the patient. Calcium ion has little or no effect. Clinical applications for this procedure have been found: (a) immediate relaxation of oxytocic-induced uterine tetany, (i) during induction of labour or management of secondary degree uterine inertia, (ii) in the third stage of labour for a separated or incarcerated placenta; (*b*) prevention of uterine tetany following the use of oxytocics; (*c*) relief of after-pains; (*d*) alleviation of essential spasmodic dysmenorrhœa; (*e*) relaxation of Bandl's ring; (*f*) relaxation of tetanically contracted uterus in *abruptio placentæ*.

The Treatment of Ureters Injured during Gynæcological Operations.

FRANCIS M. INGERSOLL AND JOE V. MIEGS (*The New England Journal of Medicine*, March, 1945) consider that accidental injury of a ureter is the most frequent serious complication of gynæcological pelvic operations. In five years at the Massachusetts General Hospital seven injuries occurred in 590 total hysterectomies and one in 170 supravaginal hysterectomies. The authors review the principles of treatment and indicate the various methods that may be adopted. They conclude that it is essential that the damage be recognized at once. Kidney function should, if possible, be preserved. All ureteral anastomoses should be performed end to end over a ureteral catheter. Bilateral ligation is best treated by bilateral nephrostomy. Uretero-vaginal fistulæ that do not close spontaneously should be repaired either by ureteral transplant to the bladder or bowel or by nephrectomy.

OBSTETRICS.

The Work of Obstetric Labour and its Significance in Heart Disease.

I. SAMPSON, E. M. ROSE AND R. QUINN (*American Journal of Obstetrics and Gynecology*, June, 1945), writing from the Department of Obstetrics of the University of California, discuss the results obtained from observations carried out on 42 patients of the work of obstetric labour and its significance in heart disease. Woodbury Hamilton and Torpin have recently attempted to measure the amount of work done during the first stage of labour. Their most interesting conclusion was that bearing down forces of the abdominal and chest muscles were not expulsive before cervical dilatation, but materially diminished the maternal placental blood pressure even to zero. The technique used in the present study was to measure the amount of oxygen utilized, and it was noted that when secondary powers were used there was a marked rise. The authors found that the work of labour in both *primipara* and *multipara* is the equivalent of mild to moderately heavy physical labour, the degree of which cannot always be predicted; any complications may cause a considerable increase. This physical work may be more than is considered safe by clinicians for life or for maintenance of maximum function of cardiac patients in the ordinary routine of life. Cæsarean section, of which one case is reported, does not carry the dangers of this unpredictable load of physical work, although post-partum cardiac death is reported to occur after this procedure for reasons as yet undetermined.

Recent Developments in Iso-Immunization by the Rh Factor.

P. LEVINE (*American Journal of Obstetrics and Gynecology*, June, 1945) simplifies the latest work on iso-immunization by the Rh factor and gives a workable basis for the clinician and obstetrician. It has been shown that *erythroblastosis fœtalis* is the result of prolonged intrauterine reaction of maternal immune agglutinins and susceptible fœtal blood. In at least 90% of these cases the mother's blood is Rh-negative and that of the father is Rh-positive, and in the remaining 10% the mothers, who have Rh-positive blood, also have finer differences of the Rh complex as well as several other blood factors besides the Rh factor. The presence of anti-Rh agglutinins, which is direct proof of immunization, has been found in only 50%. This difficulty has been met by Race and Wiener, who have independently shown that mothers with Rh-negative blood may possess antibodies which unite and coat the surface of Rh-positive blood cells without inducing the visible effect of agglutinin. From a practical point of view, although there are four types of Rh blood, it is better for the clinician to consider only the clinically important variety which gives 85% reaction. The Rh factor varies in different races, and it occurs in less than 1% of Chinese. Until all varieties of anti-Rh serum become generally available to hospital laboratories, an occasional intra-group transfusion accident may be expected. The rule in these cases is to select donors of the same subtype. The erythroblastotic infants of the rare mothers who have Rh-positive blood should receive transfusions of compatible blood of the same subtype as that of the mother. In general, if the mother's blood is incompatible, it may be used for an affected infant provided that the plasma is removed and replaced with saline solution and compatible plasma. It has become clear in the last three years that once a mother with Rh-negative blood is immunized either by transfusion or by pregnancy, she must be considered as remaining immunized for the rest of her life. One woman, eleven years after giving birth to an erythroblastotic infant, tolerated a transfusion of Rh-positive blood, but suffered from anuria with the second; another woman was prevented from having a child by receiving repeated transfusions of blood when she was six years old. So it behoves all female children to be tested before they are given repeated transfusions. Iso-immunization is probably the result of unbelievably small amounts of fœtal cells gaining access to the maternal circulation. The outlook for women with Rh-negative blood is better today, as it is possible to prevent iso-immunization by the transfusion of Rh-positive blood; the pregnant woman with Rh-negative blood should be tested periodically to detect the earliest onset of iso-immunization. Generally the failure to detect it gives a good prognosis; if iso-immunization is increasing, induction of labour with immediate transfusion offers a chance of the child's survival. No further pregnancies should be recommended until after the disappearance of all residual agglutinins or incomplete antibodies produced in the previous pregnancy. In all cases complete blood studies of mother, father and surviving children, if any, should be carried out, to determine the genotype of the father—whether he is homozygous or heterozygous.

Continued Thiourea Treatment and the Offspring of the Rat.

E. D. GOLDSMITH, A. S. GORDON AND H. A. CHARIPPER (*American Journal of Obstetrics and Gynecology*, February, 1945) record the results of their investigations on continued thiourea treatment in pregnancy and its effect on the development of the offspring of the rat. They conclude that the feeding of pregnant rats with food containing 0·5% of thiourea causes activation and hyperplasia of the thyreoid gland and retardation of the growth of the offspring. The thiourea may be transmitted directly through the food, or indirectly through the placenta or through the milk, and the authors advise caution in the use of thiourea or thiouracil therapy for pregnant patients suffering from hyperthyreoidism.

The Action of Veratrone on Blood Pressure, Urine Volume and Urea Clearance in the Toxæmias of Pregnancy.

J. R. WILLSON (*American Journal of Obstetrics and Gynecology*, May, 1945) reports the action of veratrone on blood pressure, urine volume and urea clearance in the toxæmias of pregnancy. It was found that the administration of the drug was followed by a slowing of the pulse and a pronounced fall in blood pressure, but that this was associated with a diminution of urinary output even to absolute anuria; the drug also caused a diminution of the figures obtained by the urea clearance test. Both these phenomena were more pronounced in preeclampsia.

British Medical Association News.

NOTICE.

THE General Secretary of the Federal Council of the British Medical Association in Australia has announced that the following medical practitioners have been released from full-time duty with His Majesty's Forces and have resumed civil practice as from the dates mentioned:

Dr. Mervyn Thomas, 15, The Esplanade, Manly (June 1, 1945).
Dr. Garnet Halloran, "Harley", 143, Macquarie Street, Sydney (September 17, 1945).
Dr. N. H. Meacle, 233, Macquarie Street, Sydney (September 10, 1945).
Dr. Russell Howard, 16, Collins Street, Melbourne (September 10, 1945).
Dr. J. W. Ralston, "Wickham House", Wickham Terrace, Brisbane, B.17.
Dr. R. T. Binns, 188, North Terrace, Adelaide (August 30, 1945).
Dr. E. W. Ferguson, 53, Hanbury Street, Mayfield, New South Wales (October 21, 1945).
Dr. T. N. Bolger, 280, Bronte Road, Waverley, New South Wales (October 22, 1945).
Dr. D. C. Howle, Peel Street, Tamworth, New South Wales (July 1, 1945).
Dr. Garth May, 115, Logan Road, South Brisbane, Queensland (October 5, 1945).
Dr. S. A. McDonnell, St. George, Queensland (October 12, 1945).
Dr. L. G. Hill, Eagle Terrace, Sandgate, Brisbane (November 12, 1945).

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held on June 1, 1945, at the Institute of Medical and Veterinary Science, Adelaide.

Carotenoids and Sex Determination.

PROFESSOR J. G. WOOD summarized some overseas work on carotenoids and sex determination. He referred briefly to the structure of carotenoids and pointed out biochemical differences among the algæ, each group being characterized by specific xanthophylls. Only the green algæ contained the xanthophylls characteristic of the higher plants. Professor Wood then referred to the role of carotenoids in non-hereditary sex determination, and gave an account of the work of Moewus and his colleagues on the effect of crocin and its breakdown products in determining motility, conjugation and sex in gametes of *Chlamydomonas*.

Heat and the Virus of Spotted Wilt.

DR. R. J. BEST read a paper on "Some Effects of Heat on the Virus of Tomato Spotted Wilt". Dr. Best found that the thermal death-point of the virus *in vitro* was 46° C. when pH value and redox potential were maintained at a favourable level for the virus. The rate of inactivation when followed in detail was found to be logarithmic; this enabled the effect of heat on the virus to be expressed in terms of a half-life period at a particular temperature. This was suggested as a more useful characteristic of a virus than the thermal death-point. The half-life period for tomato spotted wilt virus (at pH 7) was twenty minutes at 35° C. Experiments on the effect of heat on the virus *in vivo* had shown that exposure of infected tomato plants to a temperature of 40° C. for twenty-fours hours inactivated 97% of the virus.

Moulds and other Foreign Substances in the Lungs.

PROFESSOR J. B. CLELAND gave an account of moulds and other foreign substances in the lungs. He said that the finding of hyphæ of one of the higher fungi in the lungs of a girl, aged five years, who had died from pneumonia at the Children's Hospital, Adelaide, had focused attention on these cases. He had recently had an opportunity of seeing sections from two similar cases in the army; one patient was an aboriginal. A year before, a man, aged seventy-four years, had been found to have some small, crusty-looking white specks on the wall of a bronchiectatic cavity. When sections of these were examined, they proved to be little sclerotia of compacted hyphæ. In 1931 Professor Cleland had described a case in a woman, aged twenty-three years, in whom a large, compacted mass of felted mycelium, about three inches long and one and three-quarter inches wide, was found lying free in a large cavity, the patient having died from hæmoptysis. Quite recently Dr. J. B. Thiersch had shown him a specimen from a similar case in which a large felted mass again lay free in a cavity. In 1924 Professor Cleland had described a case of aspergillosis of the pleura with sclerotium formation (THE MEDICAL JOURNAL OF AUSTRALIA, June 28, 1924).

Correspondence.

AUSTRALIAN MEDICAL HISTORY OF THE WAR OF 1939–1945.

SIR: The termination of a state of war has accentuated the need for rapid assembling of material for the "Medical History". Much useful material of a professional kind has already been published in your pages, and in addition a number of service medical officers have been asked to contribute articles and information for the specific use of the history. Others will be similarly approached as opportunity offers.

It is to be hoped that such contributions will be promptly made, but there is much valuable material which is less easy to tap. It exists in notes, in letters, in diaries, and in the memories of those who will soon be engaged in competitive striving for a living if they are not doing so already. Time is short and so is memory. I write to ask your help in broadcasting an appeal to any medical officers of this war who may be able to give information about medically interesting aspects of their service experiences.

A medical history is concerned chiefly with problems and their solution, and in such matters it is important to know the personal views of the men in the field. Even material which is not strictly official is informative and useful. It is naturally treated as confidential and would not be used without consent.

The obligation is laid upon us to present an accurate account of these tremendous years, and we owe it to posterity to record fully and clearly our difficulties and our failures as well as our successes. Any contribution, however modest in the eyes of its author, may assist the Medical Editor in arriving at truth.

The directors of the medical services of navy, army and air force permit me to say that they are agreeable that their officers cooperate with the medical historian in this way.

Yours, etc.,

ALLAN S. WALKER,
Medical War Historian.

262, Domain Road,
South Yarra, S.E.1,
Victoria.
September 5, 1945.

SYSTEMIC ADMINISTRATION OF PENICILLIN.

SIR: Those called upon to administer penicillin continuously to a large number of cases will hasten to compliment Surgeon Lieutenant-Commander Dow and Surgeon Lieutenant Commander Martin on their contribution (THE MEDICAL JOURNAL OF AUSTRALIA of July 28, 1945). It is to be regretted, however, that no information relative to the extent of the trial has been afforded. The pitfalls of the method in our hands are such as to require special mention. In this tented hospital in the tropics we have administered 600 million units of penicillin by the continuous intramuscular method to 482 patients involving 988 separate administrations. Our apparatus has been improvised here upon the pattern recommended by the Medical Directorate, Australian Army Medical Corps. An ordinary intravenous apparatus is adapted to the very slow drip by inserting an intratubal hypodermic needle which projects into the dripper. It requires very little attention and is ideal where twenty or thirty ward administrations are proceeding simultaneously. Two hundred thousand units are administered every forty-eight hours in 250 millilitres of saline. In some cases the needle has remained *in situ* in the thigh for a week. In 12% of our administrations the thigh became reddened and tender.

but settled rapidly with fomenting, the needle being reinserted in another site. In our opinion it is important to use only saline prepared for intravenous use ("Soluvac"). Reactions with pyrexia up to 102° F. are not unusual here in the tropics during the administration. Improperly immobilized needles lead to needle track infections, of which eight cases occurred in our series. Four of these progressed to suppuration, the pus being sterile in two and yielding Gram-negative organisms in two (*Pseudomonas pyocyaneus, Bacillus coli*). These complications therefore appear in about 1% of the administrations.

Our series was interesting in that almost all patients had previously been treated for two to seven days with intermittent penicillin injections. They preferred the continuous intramuscular method because it did not preclude rest—a not unimportant consideration in the wounded.

Our observations suggest that even here in the tropics the deterioration of penicillin at room temperature is very low and less than 50% at the sixth day. A forty-eight hour dosage period was therefore employed. (The penicillin assays were performed by Captain Eric French, Australian Army Medical Corps.)

It is our firm opinion that, until oral administration of penicillin is perfected, the continuous intramuscular route is at the moment the best method of penicillin administration over a long period. More particularly is this so if careful skin sterilization at the site of insertion and correct immobilization of the needle are ensured to minimize complications.

Yours, etc.,

K. W. STARR,
Lieutenant-Colonel, Australian Army Medical Corps.

Netherlands East Indies,
August 28, 1945.

EXPERIENCES, DANGERS AND SEQUELÆ OF SULPHONAMIDE ADMINISTRATION.

SIR: I was interested in the paper by Dr. R. G. S. Harris in THE MEDICAL JOURNAL OF AUSTRALIA, June 30, 1945, page 653. I would like to comment on certain statements made by him regarding causes and treatment of anuria, which sometimes follows administration of sulphonamides, and also to call attention to the fact that crystalluria occurs with sufficient frequency, quite apart from the administration of the sulphonamides, as to cause confusion in the diagnosis of these two conditions. In December, 1940, I contributed an article to THE MEDICAL JOURNAL OF AUSTRALIA entitled "A Critical Review of So-Called Cases of Reflex Anuria", and quoted ten cases which had been reported as reflex anuria. I put forward the theory that these were cases of anuria due to bilateral ureteral blockage by an agglomeration of crystals, which at the time I referred to as calculi. These cases occurred before sulphonamides came into general use, so that none of them had had any of these drugs. I came to the conclusion that the probable nature of the crystals was either calcium oxalate or uric acid. The reported occurrence of these cases after operations, I thought, was due to the increased tendency to deposition of crystals which would occur in the highly concentrated urine resulting from dehydration of the patient frequently associated with an operation. There is little doubt that this theory has been substantiated, and so these cases are very similar to those following the administration of sulphonamides, a fact which I mentioned in my paper on the subject; the only difference being in the nature of the crystals. Since my original paper was written and after seeing the many cases which have been reported following upon the administration of the sulphonamides, I realize that the anuria in most of the cases quoted by me was due to aggregations of crystals which are not really calculi, but are the first stage in the process of calculus formation. This does not affect the main thesis which I advocated in my paper, but it does afford a simpler explanation of the successful result of the treatment which I recommended for these cases. An aggregation of crystals would produce the same symptoms as a calculus, but would be more readily dissolved or mechanically displaced.

Dr. Rose in an article in THE MEDICAL JOURNAL OF AUSTRALIA of June 2, 1945, has reported a large number of patients with crystalluria and urinary calculi occurring in tropical climates. Many of these patients had anuria and none had had any of the sulphonamides.

To obtain some idea of the frequency of renal trouble following upon the administration of sulphonamides, I studied the case histories of all the cases of pneumonia and of meningitis which had been treated in the Royal Alexandra Hospital for Children during the twelve-month period between July, 1943 and 1944, a year during which sulphapyridine had been mainly used. I found that there had been two hundred and eighty cases of pneumonia and sixty-one cases of meningitis. All the cases of pneumonia and all except five of the cases of meningitis had been treated with appropriate doses of sulphapyridine. Out of these three hundred and forty-one cases, there had been nine cases where gross hæmaturia had been reported, all of which rapidly cleared up after discontinuing the drug and giving increased fluids. Although oliguria had been reported in some of the cases, none had had anuria. With regard to the site of the blockage causing anuria or rather suppression of urine, although there is plenty of evidence that blockage of some of the renal tubules, as well as of the ureters, can be produced by administering large doses of one of the sulpha drugs, mainly sulphathiazole, to rats; and although by special staining methods crystals can sometimes be demonstrated in the tubules of the kidney in persons who have died after an unrelieved anuria, following upon the administration of one of the sulphonamides, I can find no conclusive evidence in the literature of any case of anuria which has been proved to be due to a complete blockage of all the renal tubules by crystals. Even in those rare cases where the kidney has been found to be almost choked with amorphous or crystalline deposit there was oliguria but not anuria. With regard to the treatment of these cases, this resolves itself into the removal of the obstruction by the simplest and most effective method possible. The administration of copious alkaline fluid is the first method of treatment. The passage of ureteral catheters may be attempted, but, as pointed out by Dr. Harris, this is not always possible. Massage of the lower ends of the ureters *per rectum* in order to dislodge the obstruction has been successfully used in some cases. Massage of the kidneys with the idea of raising the intrapelvic pressure and so dislodging ureteral crystals may be effective. If these measures fail, one should by-pass the obstruction by nephrostomy or pyelostomy, and as the superadded spasm and congestion of the ureters subsides, any accumulation of crystals or any small calculi will usually pass along into the bladder. Other methods of treatment mentioned by Dr. Harris, such as heat in the form of "inductothermy", or infra-red rays, the use of the shock cradle and decapsulation of the kidneys, will not be necessary once the obstruction in the ureters has been overcome.

Yours, etc.,

P. L. HIPSLEY.

141, Macquarie Street,
Sydney,
September 5, 1945.

POVERTY, HOUSING AND HEALTH.

SIR: Dr. J. Brown, in his letter to the journal of September 1, 1945, raises so many important questions, covering such a large field of inquiry, that a full reply would need a book, so it is possible to comment on only a few points.

I think it is unfair to accuse sociologists of "holding up medical practitioners as examples of ineptitude and indolence". Their complaint is that the medical profession, acting as an organized body, has not continually used its influence to inform the public of the dreadful effects of poverty in causing crime, disease and death.

Certainly we find in medical textbooks that poverty and slums are included among the causes of tuberculosis; but it is not enough to have a passing mention in a textbook; the professional conscience cannot be clear until we, acting as a body through our associations, have taken every possible step to drive deeply into the public consciousness the dreadful price poverty exacts from a nation. The profession is politically weak because it has never tried to arm the public with facts; and to use that armed intelligence against this evil, which is now an anachronism.

Only the socially ignorant question the fact of the poverty of the majority of our population. In the last pre-war year 80% of our workers were earning £5 a week or less; after a short period of higher wages the majority is again earning £5 a week or less. Of a basic wage of £5, thirty-five shillings are allowed for food for a man and his wife and one child. I invite Dr. Brown, and all who like to imagine, for the ease of their consciences, that the Australian workers are comfortably off, to limit themselves and their families to that rate for, say, three months. And let them remember that there is no fund for contingencies in the basic wage; it is all neatly parcelled out—so much for rent, so much for clothing, so much for union and lodge fees *et cetera*, so that any

extra expenses, such as prolonged sickness, replacements of furniture and cooking utensils, a holiday trip, a visit to a symphony concert or a humble talkie, must be paid for by economizing on the food bill. Then let them say whether the average worker can, under present conditions, fairly be expected to make any contribution to a social security fund.

Dr. Brown's belief "that there is a mathematical relationship between the total effective work done and the standard of living in a community" suggests an innocently benevolent view of capitalism. A survey in England showed that from 1932 to 1936 the average increase in production per worker was 18·6%. In some cases the rise was enormous; for example, pig-iron 55%, cars and aircraft 43%, artificial silk 64%. On Dr. Brown's thesis all that increased productivity would have been divided among the whole community. Was it? No. Wages and prices remained substantially the same, and "profits" took the lot.

The transfer of slum dwellers that Mr. Burt quoted occurred in the Stockton-on-Tees project, and the number of people moved was 710. The number left in the original slums was 1,298, and the period covered by the investigation was four years. The death rate was standardized, and McGonigal and Kirby, who are experienced medical officers of health, gave it as their considered opinion that there was no explanation for the rise in death rate except the lessened amount of money that could be spent on food.

Yours, etc.,

E. P. DARK.

Katoomba,
New South Wales,
September 7, 1945.

SIR: Dr. J. Brown in his criticism of Mr. W. Oswald Burt's article on poverty, housing and health in turn calls for criticism. In his letter he makes a very true, and for these days, a very topical statement, "our aim should be to provide work for all". However, he is a defeatist in saying: "If little political action has resulted, can medical practitioners be blamed for that? Politically they are non-entities, having negligible voting strength." Though in voting strength we may be "negligible", no one can deny that the influence of the profession in the community is very considerable. If our influence was used in an organized way to further such laudable aims as full employment, and better housing conditions, and higher wages, we would greatly benefit public health. The go-slow policy which Dr. Brown condemns, I think, is adopted by workers because they realize that harder work means larger profits to the employers and little real benefit to themselves. Higher wages, better housing, better nutrition and full employment would result in greater industrial output as well as better health.

Yours, etc.,

DAVID MOORE.

Balmain and District Hospital,
Booth Street,
Balmain.
September 7, 1945.

A MEMORIAL TO THE LATE MISS A. M. KELLETT.

SIR: The New South Wales Branch of the Australian Nursing Federation (Australasian Trained Nurses Association) has decided to found a memorial to the late Miss A. M. Kellett in recognition of the splendid and unselfish work which she did for nursing throughout her long career. The memorial will take the form of a fund for the endowment of a prize to be given each year to the candidate who obtains the highest marks in the final examinations conducted by the Nurses' Registration Board in New South Wales. A committee of management consisting of Miss C. Gilchrist, Miss R. A. Kirkcaldie, Miss H. B. Hetherington, Miss E. Pidgeon and Miss E. P. Evans has been appointed.

We are sure you will agree that no more appropriate tribute could be paid to Miss Kellett, whose constant endeavour was the improvement of the standard and the enlargement of the scope of nursing as well as the furtherance of the ideals and aspirations of the noble profession to which her life was dedicated.

The nature of the prize will necessarily depend upon the amount subscribed, but a favoured suggestion is that it take the form of an enamelled silver medallion suitably inscribed, together with a gift as funds permit.

Donations to this fund are invited and should be forwarded to the undersigned at Manchester Unity Building, 185, Elizabeth Street, Sydney.

Yours, etc.,

E. P. EVANS,
General Secretary.

Australian Nursing Federation,
185, Elizabeth Street,
Sydney.
September 7, 1945.

REPATRIATION DEPARTMENT AND EX-SERVICE PERSONNEL.

SIR: I refer to the letter of Dr. D. R. W. Cowan, of Adelaide, in the issue of September 1, 1945, in which reference is made to previous correspondence in November, 1944.

Once again Dr. Cowan is incorrect in his statement. In my reply to his original letter I pointed out that it was not nor had it ever been the policy of the Repatriation Department to insist on the finding of tubercle bacilli before a diagnosis of pulmonary tuberculosis was made.

He now states that if such is not the policy it certainly is the practice commonly adopted. I have now to add that this statement is incorrect, and that he could have no evidence to support it.

Yours, etc.,

KENNETH SMITH,
Principal Medical Officer.

Repatriation Commission,
314, Collins Street,
Melbourne.
September 10, 1945.

THE PREVENTION OF FRACTURES DURING SHOCK THERAPY.

SIR: A full reply to Dr. A. R. Hamilton's letter, published in the correspondence column of your journal of August 25, 1945, is not possible within the small space devoted to that column. However, an excellent summary of the whole position regarding skeletal complications of convulsion therapy, with full references, is published in the January, 1944, edition of *The Journal of Mental Science*, pages 442 to 447. There is another article on the same subject in the November, 1944, *Bulletin of the New York Academy of Medicine*.

It would appear that orthopædists in this country are not aware that immobilization and splinting apparatus are not now considered necessary in the treatment of minor compression fractures of dorsal vertebræ due to this form of trauma.

To answer Dr. Hamilton's questions briefly:

1. Psychiatrists generally think that restrictive apparatus and restriction of movement are not only ineffective but dangerous.
2. Curare, whilst effective in "softening" the convulsion, adds a danger to life, to a procedure that is not in itself dangerous to life.
3. The best posture is that in which the patient lies with the knees and hips semiflexed, the dorsal spine extended and the arms abducted and externally rotated.

It may be sufficient to add that psychiatrists in all countries have been fully aware of the hazards associated with this form of therapy and have sought expert orthopædic advice to enable them to produce the best possible technique for the avoidance of these dangers.

Yours, etc.,

CEDRIC SWANTON.

British Medical Association House,
135, Macquarie Street,
Sydney.
September 12, 1945.

INTERVERTEBRAL DISK DISEASE: NEW SYNDROMES AND CONCEPTS.

SIR: Dr. Douglas Miller, in his letter of August 31 (THE MEDICAL JOURNAL OF AUSTRALIA, September 15, page 351), rightly takes exception to a badly worded statement in my article—a statement which has suffered in several processes of condensation. This I regret. May I therefore amplify.

Certain deformities are not "primarily" due to muscular spasm. They are "primarily" due to the protrusion of a piece of disk which prevents the vertebræ from being extended or from being flexed to the affected side. These movements, by compressing the protrusion, cause pain. This pain may and does lead to a secondary muscular spasm.

Some of the localized deformities cannot be produced by muscular action and are in fact caused by the mechanical effect of a protrusion. The deformity "may" then persist under adequate anæsthesia. I have in my possession X rays revealing widening of the intervertebral space before operation and narrowing of the space after operation. In this case the vertebræ were wedged apart by a disk fragment and the deformity disappeared immediately this part of the disk was removed. Dr. F. J. Clark and I have seen other similar cases. Dr. Miller states that he has not seen this, and this must be because he has not operated on early cases.

I quite agree with Dr. Miller that part of the disk is a little mass of soft structureless material, that is, the *nucleus pulposus*, but would not agree that protrusion of this part of the disk causes symptoms. The part of the disk which is responsible for the symptoms is the *annulus fibrosus*, which is composed of tough fibrocartilage.

The great majority of disk lesions will recover with conservative treatment. The place of surgery can therefore only be to relieve pain or disability which cannot be relieved by other methods, or to shorten a period of disability. When there is a localized deformity which cannot be produced by muscular action and which must be, and in fact is, due to mechanical wedging by a disk fragment, it is my experience that surgery may shorten the period of disability considerably and that early operation is therefore justified.

In those cases of low back pain which have been treated surgically we have had no great difficulty in diagnosing the pathological type of disk disease, nor in localizing the affected disk or disks by clinical examination, even in the absence of pain in the lower limb, of neurological signs, or of positive X-ray findings; and we have found it a very simple matter to confirm the diagnosis by the mobility test at operation.

I do not consider that all pains in the back, neck and limbs have a common denominator in the intervertebral disk, but I am convinced that many more of these pains have their origin in disk lesions than we have hitherto thought.

Dr. Miller refers to conservative and radical surgery. We have found as a result of experience that, if surgical treatment is indicated, the disk must be removed as completely as possible, but I hasten to assure Dr. Miller that, in Dr. Clark's hands, radical surgery is not destructive.

Yours, etc.,

JAMES H. YOUNG.

131, North Beach Road,
Mount Hawthorn,
Western Australia.
October 5, 1945.

CAN EPILEPSY BE CURED?

SIR: I would like to congratulate Dr. N. V. Youngman on his article, "Can Epilepsy be Cured? The Results of Treatment", in the journal for September 15, 1945. Dr. Youngman states that "epilepsy is merely a name, the name for a group of symptoms"; he also refers to "the constitutional predisposition". It is this "constitutional predisposition" to which I wish to refer. I recently attended a medical meeting at which a number of speakers discussed "allergy" in relation to various general and special departments of medical practice.

Unfortunately I missed the opening remarks by Dr. George Taylor on the condition of allergy itself. Several speakers referred to the feature of heredity, but in the main such suggestions for treatment which were offered dealt with the treatment of various symptomatic manifestations of the condition and not with the basic cause. This could only be expected, for the basic cause of the condition is unknown, being some constitutional disorder of a biochemical, endocrine *et cetera*, nature, or "constitutional predisposition", as stated by Dr. Youngman in connexion with epilepsy.

At the meeting referred to I had reluctantly agreed to speak on allergy in relation to conditions of the ear, nose and throat. I found great difficulty in doing this, for allergy in relation to the ear, nose and throat cannot be divorced from general medicine, and except for a few therapeutic measures against a few symptoms such as hay fever is not a question for local treatment.

The conditions or symptoms which are due to this familial constitutional dysfunction are many—asthma, hay fever, "indigestion", "nerves", migraine, Ménière's syndrome, eczema, urticaria, epilepsy and many others.

In carefully taken case histories including "family history", also details of the patient's actual living and working conditions, family discords *et cetera*, this familial nature is more and more emphasized.

The case of a young married woman who consulted me and gave an almost perfect description of Ménière's syndrome as her complaint is a good example. Her sister had had bilateral, radical antrum operation, her eldest child was under treatment for epilepsy, she herself also suffered from "nerves".

I would once again stress the "allergic" origin of most of what is called "sinusitis" and bronchiectasis. These two conditions are the same, but situated in different sites, both are congenital in origin, and the subsequent course is influenced by various factors including infection. Dr. Youngman refers to the "time-honoured soil seed relationship", and it is the "soil" factor that is so important and so neglected. The initial conditions are very frequently overlooked and can only be diagnosed at present by combining careful case-taking, including "family history", with careful physical examination.

Many times I have diagnosed "bronchiectasis" by ear when the patient was in another room. Physical examination of the lungs by an expert combined with X ray has resulted in a diagnosis that the chest is clear, but subsequent developments tell a different story. Also with asthma. The manifestations of this disorder are responsible for an enormous amount of suffering and disability and the loss of much in human work and endeavour.

I wish to again emphasize the need for organized and government aided research in the subject of biochemistry, which must be collaborated with the work of practising clinicians. It is even possible that in this work may be the solution of the problem of malignant disease.

Yours, etc.,

ERNEST CULPIN.

Wickham Terrace,
Brisbane,
Undated.

Australasian Medical Publishing Company, Limited.

ANNUAL MEETING.

THE annual meeting of the Australasian Medical Publishing Company, Limited, was held at The Printing House, Seamer Street, Glebe, New South Wales, on October 2, 1945, DR. T. W. LIPSCOMB, the chairman, in the chair.

Directors' Report.

The report of the directors of the company was as follows:

The directors submit their report for the twelve months ended June 30, 1945, together with the balance sheet as at June 30, 1945, and the profit and loss account for the twelve months ended June 30, 1945.

During the year THE MEDICAL JOURNAL OF AUSTRALIA published articles which covered a wide range of subjects and the standard of previous years was maintained. Arrangements have been made to publish additional pages of reading matter in the forthcoming year.

The output of the printing and publishing department may be regarded as satisfactory. It was of the usual high quality and was mainly of an educational and scientific nature. The company is still faced with difficulties in regard to manpower and paper, but, with the cessation of hostilities, it is hoped that the position will improve in the early future.

The directors have under consideration a plan to extend The Printing House. The building is at present overcrowded, and when the possibility of further expansion of the company's activities and the present inadequate accommodation for the printing staff are taken into consideration, the directors feel that the plan should be put into operation as soon as possible.

Depreciation amounting to £853 was written off during the year and an amount of £1,757 has been provided for taxation. The company's reserves are used in the business, and we consider the state of the company's affairs is satisfactory.

Provision has been made for the payment of debenture interest for the year ended June 30, 1945.

Dr. D. Gifford Croll and Dr. A. M. Davidson retire from office by rotation in accordance with the Articles of Association (Article 39). They are eligible and present themselves for reelection.

T. W. LIPSCOMB,
Chairman.

September 1, 1945.

Election of Directors.

Dr. D. Gifford Croll and Dr. A. M. Davidson were reelected to the Board of Directors.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 161 and 166, of August 23 and 30, 1945.

PERMANENT NAVAL FORCES OF THE COMMONWEALTH (SEA-GOING FORCES).

Transfer to Retired List.—Surgeon Lieutenant-Commander (D) Sydney Joseph Leon Abrahams is transferred to the Retired List, dated 13th August, 1945.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

The grants of the acting rank of Wing Commander to the following Temporary Squadron Leaders are terminated upon their ceasing to occupy Wing Commander posts with effect from the dates indicated: H. A. A. Altmann (251766), 15th June, 1945, W. Deane-Butcher (261286), 18th June, 1945.

The appointment of Temporary Squadron Leader A. L. Webb (267631) (part-time) is terminated at his own request with effect from 30th June, 1945.

The appointment of Temporary Flight Lieutenant R. A. Duval (267693) is terminated at his own request with effect from 9th October, 1944.

The notification concerning Temporary Flight Lieutenant R. A. Duval (267693), appearing in the *Commonwealth of Australia Gazette*, No. 237, dated 30th November, 1944, is cancelled.

Reserve: Medical Branch.

The following officers are transferred from the Active List with effect from the dates indicated: (Temporary Squadron Leaders) M. Morris (251267), 3rd July, 1945, F. T. Humphrey (261369), 5th July, 1945, Temporary Flight Lieutenant F. L. Davey (251236), 3rd July, 1945.—(Ex. Min. No. 209—Approved 22nd August, 1945.)

Temporary Squadron Leader J. H. Deakin (261767) is transferred from the Active List with effect from 26th June, 1945.

Temporary Squadron Leader M. H. B. Robinson (251257) is transferred from the Active List with effect from 20th July, 1945.—(Ex. Min. No. 217—Approved 29th August, 1945.)

Temporary Squadron Leader H. A. Sundstrup (271256) is transferred from the Active List with effect from 12th July, 1945.

CASUALTIES.

ACCORDING to the casualty list received on September 4, 1945, Colonel J. E. Gillespie, East Malvern, Victoria, has been removed from the "dangerously ill" list.

According to casualty lists received on September 12, 1945, Lieutenant-Colonel R. G. Williams, North Cottesloe, Western Australia, has been removed from the "seriously ill" list and Colonel E. R. White and Lieutenant-Colonel A. E. Coates, both of Toorak, Victoria, who were previously reported prisoners of war, are now reported "not prisoner of war".

According to the casualty list received on September 13, 1945, Major H. H. Eddey, Footscray, Victoria, and Captain I. C. Heinz, South Yarra, Victoria, who were previously reported as prisoners of war, are now reported "not prisoner of war".

According to the casualty lists received on September 17, 1945, Major T. P. Crankshaw, of Malvern, Victoria, previously reported prisoner of war, is now reported "not prisoner of war", and Captain M. M. Brown, of Hobart, Tasmania, previously reported prisoner of war, is now reported "missing, believed deceased".

According to the casualty lists received on September 20, 1945, the undermentioned, previously reported as prisoners of war, are now reported "alive in Siam". Victoria: Major H. L. Andrews, Murchison; Captain E. B. Dreverman, Armadale; Captain J. L. Frew, Toorak. Tasmania: Captain T. G. H. Hogg, Launceston.

According to the casualty lists received on September 20, 1945, Lieutenant-Colonel W. A. Bye, of Sydney, who was previously reported to be a prisoner of war, is now reported "recovered".

According to the casualty lists received on September 20, 1945, the undermentioned are now reported "recovered Singapore": Major K. B. Burnside, Toorak, Victoria; Major J. F. J. Cade, Bundoora, Victoria; Major I. T. Cameron, Croydon, Victoria; Captain A. R. Bush, Melbourne; Captain J. P. Catchlove, Elwood, Victoria; Captain G. F. Braby, Maribyrnong, Victoria.

According to the casualty lists received on September 20, 1945, Captain P. N. O'Donnell, of Melbourne, is reported "recovered Manilla".

According to the casualty lists received on September 24, 1945, Major R. Dick, Balgowlah, and Major K. J. Fagan, Bookham, New South Wales, previously reported prisoners of war, are now reported "recovered".

According to the casualty lists received on September 24, 1945, Captain G. J. Horder, Malvern, Victoria, is reported "recovered Manilla".

According to the casualty lists received on September 24, 1945, Major R. G. M. Forsyth, Burnley, Victoria, and Captain W. Dixon, Elwood, Victoria, are reported "recovered Singapore".

According to the casualty lists received on September 24, 1945, Captain G. M. Crabbe, New Norfolk, Tasmania, previously reported prisoner of war, is now reported "recovered Borneo".

According to the casualty lists received on September 24, 1945, Captain J. E. R. Clarke, Brisbane, Queensland, previously reported prisoner of war, is now reported "located Siam".

According to the casualty lists received on September 27, 1945, the following, who were previously reported prisoners of war, are now reported "recovered": Captain D. J. Brennan, Sydney; Major F. P. C. Claffy, Sydney; Captain P. I. A. Hendry, Strathfield; Captain T. Le-Gay-Brereton, Turramurra; Major R. E. Maffey, Singleton; Captain F. H. Mills, Bellinger; Captain F. F. Elliott, Merewether; Captain R. A. McDonald, Eastwood; Major H. Rayson, Manilla; Captain F. R. Reid, Maroubra; Lieutenant-Colonel E. MacA. Sheppard, Adamstown; Major B. L. W. Clarke, Brisbane; Captain C. R. Boyce, Toowoomba.

According to the casualty lists received on September 27, 1945, Major G. N. S. Campbell, Vaucluse, New South Wales, previously reported prisoner of war, is now reported "died whilst prisoner of war".

According to the casualty lists received on September 27, 1945, the following, who were previously reported prisoners of war, are now reported "alive in Siam": Captain J. T. Finimore, Ipswich, Queensland; Captain R. W. Greville, Hawthorn, Victoria; Captain E. J. I. Watt, Launceston, Tasmania.

According to the casualty lists received on September 27, 1945, Captain F. W. S. Finch, Mount Lawley, Western Australia, who was previously reported prisoner of war, is now reported "recovered—date not given at Singapore".

According to the casualty lists received on September 27, 1945, Captain J. E. Throssell, Mount Hawthorn, Western Australia, who was previously reported prisoner of war, is now reported "recovered Borneo".

According to the casualty lists received on September 27, 1945, Major J. F. Akeroyd, Frankston, Victoria, is reported "recovered and placed on seriously ill list".

According to the casualty lists received on October 2, 1945, Captain D. C. C. Hinder, Gordon, New South Wales, and Captain C. R. R. Huxtable, Southport, Queensland, who were previously reported prisoners of war, are now reported "recovered".

According to the casualty lists received on October 4, 1945, the undermentioned, who were previously reported to be prisoners of war, are now reported "recovered": Captain A. K. Barrett, Rose Bay, New South Wales; Captain J. P. Higgin, Cronulla, New South Wales; Captain L. O. S. Poidevin, Scone, New South Wales; Captain S. E. J.

Robertson, Bellevue Hill, New South Wales; Captain R. G. V. Parker, Rose Bay, New South Wales; Captain C. P. Juttner, Woodside, South Australia; Captain A. J. M. White, Hobart, Tasmania.

According to the casualty list received on October 4, 1945, Captain W. Aitken, Coburg, Victoria, is reported "recovered—area unstated".

According to the casualty lists received on October 4, 1945, Captain V. G. Bristow, Heidelberg, Victoria, is reported "recovered Japan".

According to the casualty list received on October 4, 1945, Major A. F. Hobbs, Fitzroy, South Australia, is now reported "repatriated".

According to the casualty lists received on October 18, 1945, Major E. L. Corlette, Orange, New South Wales, Major A. A. Moon, Neutral Bay, New South Wales, and Major P. F. Murphy, Bellevue Hill, New South Wales, who were previously reported as being alive in Siam, are now reported "recovered".

According to the casualty lists received on October 18, 1945, Captain G. D. Cumming, Manly, New South Wales, and Captain S. T. Simpson, Brisbane, who were previously reported as prisoners of war, are now reported "recovered".

According to the casualty lists received on October 18, 1945, Major W. G. Pickering, Rockdale, New South Wales, is reported "placed on 'seriously ill' list".

According to the casualty lists received on October 25, 1945, Captain A. E. Ramsey, Cremorne, New South Wales, is reported as having died, and Major A. R. Hazelton, Artarmon, New South Wales, who was previously reported "alive in Siam", is now reported "recovered".

According to the casualty lists received on October 29, 1945, Major G. F. S. Davies, Camperdown, New South Wales, Major W. E. Fisher, Rose Bay, New South Wales, Lieutenant-Colonel T. Hamilton, Newcastle, New South Wales, Captain R. G. Wright, Wagga, New South Wales, and Captain E. N. Lee, New Town, Tasmania, who were previously reported as being alive in Siam, are now reported "recovered".

According to the casualty list received on October 29, 1945, Major S. Krantz, Adelaide, South Australia, is now reported "repatriated".

According to the casualty lists received on November 12, 1945, Captain J. B. Oakeshott, Lismore, New South Wales, is reported to have died whilst a prisoner of war, and Captain E. J. Thomson, Greenbushes, Western Australia, has been placed on the "seriously ill" list.

According to the casualty lists received on November 15, 1945, Captain R. C. Hunt, Dandenong, Victoria, is reported "placed on and removed from the 'seriously ill' list".

Post-Graduate Work.

COURSE IN PÆDIATRICS AT SYDNEY.

THE New South Wales Post-Graduate Committee in Medicine desires to announce that a course in pædiatrics will be held at the Royal Alexandra Hospital for Children, Camperdown, New South Wales, from December 10 to 15, 1945. The fee for the course is £3 3s. Arrangements will be made for demobilized medical officers to attend this course under Reconstruction Training Benefits on application to the Secretary of the Committee, 131, Macquarie Street, Sydney. Telephone: BW 7483.

Australian Medical Board Proceedings.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1939 to 1940, of Queensland, as duly qualified medical practitioners:

Hayward, George Murray, M.B., B.S., 1943 (Univ. Melbourne), c.o. Leichhardt Hotel, Bolsover Street, Rockhampton.

Cranswick, Edward Harvard, M.B., B.S., 1940 (Univ. Sydney), General Hospital, Maryborough.

Mead, Kevin William, M.B., B.S., 1945 (Univ. Sydney), General Hospital, Brisbane.

Green, Ross Leonard, M.B., B.S., 1945 (Univ. Queensland), Hume Street, Boonah.

Higlett, Keith Mortimer, M.B., B.S., 1945 (Univ. Queensland), c.o. Queensland National Bank, Limited, Murgon, Queensland.

The following additional qualification has been registered:

Patterson, Mervyn Stuart, Ch.M., 1909 (Univ. Sydney), 57, East Street, Ipswich.

TASMANIA.

THE undermentioned has been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as a duly qualified medical practitioner:

Shoobridge, Michael Philip Kendal, M.B., B.S., 1945 (Univ. Melbourne), Launceston General Hospital, Tasmania.

Corrigendum.

IN the article by Professor E. R. Trethewie entitled "Anti-coagulant Effect of Perfusing Organs: Liver, Kidney and Lung", published in the issue of September 15, 1945, two errors have appeared. On page 329 in the line immediately above Table II the words "Figure I" should be "Figure II". On page 331 in the paragraph under the sub-heading "Prothrombin Content of Plasma" the words "Figure II" in the third line from the bottom of the paragraph should be "Figure I".

Obituary.

WE regret to announce the deaths of the following medical practitioners, which occurred on the dates and at the places mentioned:

Robert William Cooper, in July, 1942, lost at sea.

George Harold Guthridge, September 10, 1945, Melbourne.

David Guthrie Hunter, September 11, 1945, Eastwood, New South Wales.

James Thomas Mitchell, September 15, 1945, Willoughby, New South Wales.

Clarence Archibald Mitchell, September 21, 1945, Lane Cove, New South Wales.

St. Andrew William Logan McDowall, September 28, 1945, Warwick, Queensland.

Francis Wilfred Harlin, September 29, 1945, Goodna, Queensland.

Percy Gowan Clarke, October 25, 1945, Melbourne.

George David Harris, October 28, 1945, Renmark, South Australia.

Archibald McKillop, November 7, 1945, Sydney.

Andrew Eric Aspinall, November 10, 1945, Turramurra, New South Wales.

David Hardie, November 11, 1945, Brisbane.

Arthur Thomas Hoskins, November 14, 1945, Longford, Tasmania.

Herbert Michael Moran, November 20, 1945, Cambridge, England.

Nominations and Elections.

THE undermentioned have applied for election as members of the Tasmanian Branch of the British Medical Association:

Bean, Roy Herbert Donald, M.B., B.S., 1944 (Univ. Melbourne), New Norfolk, Tasmania.

Nolan, Philip, B.A., M.B., B.S., 1944 (Univ. Melbourne), Bothwell, Tasmania.

The undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Lebanon, Miriam, M.B., B.S., 1944 (Univ. Queensland), 5-124, Francis Street, Bondi.

Rundle, Herbert Wesley, M.B., B.S., 1945 (Univ. Sydney), The Maitland Hospital, Maitland.

Dowling, John Laidley, M.B., B.S., 1937 (Univ. Sydney), 1, Rose Bay Avenue, Bellevue Hill.

Griffiths, William James, M.B., B.S., 1942 (Univ. Sydney), Queanbeyan.

Spence, Olaf McClure, M.B., 1936 (Univ. Sydney), 102, Northwood Road, Lane Cove.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association as from July 1, 1945:

Berger, Pinkas, M.D., 1935 (Bratislava, Czechoslovakia), registered in accordance with the provisions of section 17A of the *Medical Practitioners Act*, 1938-1939, NX77177 Captain, 17, Douglas Parade, Rose Bay.

Booth, Ian Stanley, M.B., B.S., 1940 (Univ. Sydney), Captain, 2/6 Aust. General Hospital, A.I.F.

Bunn, Terence Aubrey, M.B., B.S., 1945 (Univ. Sydney), Mater Misericordiæ Hospital, Waratah, New South Wales.

Cuthbert, Antony Bevan, M.B., B.S., 1945 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Diethelm, Anton Emil, M.B., B.S., 1941 (Univ. Sydney), "Cheddington", 9, Rosemont Avenue, Woollahra.

Fraser, Allan Arthur, M.B., B.S., 1945 (Univ. Sydney), 36, Carlton Parade, Carlton.

Jones, Lloyd James, M.B., B.S., 1944 (Univ. Sydney), Prince Henry Hospital, Little Bay.

Lee, E. B., M.B., B.S., 1944 (Univ. Sydney), 22, Strickland Avenue, Lindfield.

Molesworth, Edmund James Cairns, M.B., B.S., 1939 (Univ Sydney), 6, "Carris Court", 33, William Street, Double Bay.

Perry, David Clayton, M.B., B.S., 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Harrison-Potter, Frances Del, M.B., B.S., 1945 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

Reddel, Arthur Edward McKenzie, M.B., B.S., 1941 (Univ. Sydney), 23, Goodwin Avenue, Ashfield.

Roberts, Arthur Edward, M.B., B.S., 1945 (Univ. Sydney), Melbourne Road, East Lindfield.

Roberts, Walter McPherson, M.B., B.S., 1929 (Univ. Sydney), 16, Helen Street, Merewether.

Terrey, George Russell, M.B., B.S., 1944 (Univ. Sydney), 38, Stanton Road, Mosman.

Walton, William Basil, M.B., B.S., 1937 (Univ. Sydney), 206c Victoria Road, Bellevue Hill.

Wilkinson, Richard, M.B., B.S., 1943 (Univ. Sydney), NX206366 Captain, R.R. and G.D.D., Redbank, Queensland.

Medical Appointments.

Dr. J. E. Hughes has been appointed temporary honorary assistant surgeon, Dr. J. F. Frayne honorary clinical assistant to the medical section, and Dr. R. F. West temporary medical officer in charge of the resuscitation unit, at the Royal Adelaide Hospital, South Australia.

Dr. B. Short has been appointed medical officer on probation at the Aboriginal Settlement, Palm Island, in pursuance of the provisions of *The Public Service Acts*, 1922 *to* 1924, and *The Aboriginals Preservation and Protection Act of* 1939, of Queensland.

Dr. D. T. Shortridge has been appointed resident medical Officer at the Royal Adelaide Hospital, Adelaide.

Dr. E. L. Morgan, Principal Microbiologist, has been appointed Director of Pathological Laboratories, Department of Public Health, New South Wales.

Dr. E. J. Dalyell, Assistant Microbiologist, has been appointed Assistant Director of Pathological Laboratories, Department of Public Health, New South Wales.

Dr. S. W. King, Assistant Microbiologist, has been appointed medical officer, Department of Public Health, New South Wales.

Dr. A. D. D. Pye, Dr. D. E. Trumpy and Dr. O. S. Hirschfeld have been appointed members of a committee set up to prepare a standard pharmacopœia for hospitals functioning under *The Hospitals Act*, 1936 *to* 1944, of Queensland.

Dr. H. V. Hanson, medical officer, Department of Public Health, New South Wales, has been appointed Chief Medical Referee, Government Insurance Office, Department of Public Health, New South Wales.

Dr. F. G. T. Turner has been appointed honorary clinical assistant to the dermatological section of the Royal Adelaide Hospital, Adelaide.

Diary for the Month.

DEC. 4.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
DEC. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
DEC. 5.—Victorian Branch, B.M.A.: Branch Meeting.
DEC. 5.—Western Australian Branch, B.M.A.: Council Meeting.
DEC. 6.—South Australian Branch, B.M.A.: Council Meeting.
DEC. 6.—New South Wales Branch, B.M.A.: Special Groups Committee.
DEC 11.—New South Wales Branch, B.M.A.: Medical Politics Committee.
DEC. 11.—Tasmanian Branch, B.M.A.: Ordinary Meeting.
DEC. 13.—New South Wales Branch, B.M.A.: Branch Meeting.
DEC. 14.—Queensland Branch, B.M.A.: Annual Meeting.
DEC. 17.—Victorian Branch, B.M.A.: Hospital Subcommittee.
DEC. 17.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
DEC. 18.—Victorian Branch, B.M.A.: Organization Subcommittee.
DEC. 18.—New South Wales Branch, B.M.A.: Ethics Committee.
DEC. 20.—South Australian Branch, B.M.A.: Council Meeting.
DEC. 20.—Victorian Branch, B.M.A.: Executive Meeting.
DEC. 21.—Queensland Branch, B.M.A.: Council Meeting.
DEC. 26.—Victorian Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract practice appointments in Western Australia. All Public Health Department appointments.

Editorial Notices.

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